Dissertation on

CLINICAL ANALYSIS OF PAPILLEDEMA

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

REGIONAL INSTITUTE OF OPHTHALMOLOGY
GOVTERNMENT OPHTHALMIC HOSPITAL
MADRAS MEDICAL COLLEGE
CHENNAI - 600 003.

THE TAMIL NADU
DR.M.G.R.MEDICAL UNIVERSITY,
CHENNAI – 600 032.

MARCH 2010

CERTIFICATE

This to certify that the dissertation entitled **"A CLINICAL ANALYSIS OF PAPILLEDEMA"** is a bonafide original work of **Dr.R.SIVAKALAI,** in partial fulfillment of the requirements for **M.S.Degree Branch – III (Ophthalmology)** Examination of the Tamilnadu Dr.M.G.R Medical University to be held in March 2010.

Prof.Dr. K. NAMITHA BHUVANESWARI M.S.D.O.
Prof. of ophthalmology and Head of the
Department of Pediatric Ophthalmology,
Neuro ophthalmology and Squint Services,
Regional Institute of Ophthalmology
Government Ophthalmic Hospital,
Egmore, Chennai-8.

Prof.Dr. M. RADHAKRISHNAN, M.S D.O.
Director And Superintendent,
Regional Institute of Ophthalmology
Government Ophthalmic Hospital,
Egmore, Chennai-8

Dr.J.MOHANA SUNDARAM, M.D, DNB., Ph.D.,
Dean
Madras Medical College and Government General Hospital
Chennai – 600 003.

ACKNOWLEDGEMENT

I owe my thanks to the Dean, **Dr.J.MOHANASUNDRAM, M.D., DNB., Ph.D.,** Madras Medical College and Hospital, for granting me permission to conduct this study at the Regional Institute of Ophthalmology and Government Ophthalmic Hospital attached to Madras Medical College and Hospital.

I thank the respected **Prof.Dr.M.RADHAKRISHNAN, M.S., DO** Director and Superintendent, Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College for having been very much supportive and encouraging for the conduct of this study.

I extend my sincere and heartfelt thanks to **Prof.Dr.K. NAMITHA BHUVANESWARI, M.S., D.O.,** for her invaluable guidance throughout my study.

I am grateful **Dr.P.GEETHA, M.S, D.O.**, and **Dr.B.PRAMILA,M.S.**, Asst. Professors, for their valuable guidance, support and encouragement rendered to me during the study.

I am extremely grateful to all the Assistant Professors of Neurology, Madras Medical College, for their support during the study.

I thank my study subjects who formed the back bone of the study and without whom this work would not have been possible.

Last but not the least, I thank "God, the Almighty" for being my guiding light all the way.

CONTENTS

ABBREVIATIONS

ICP	-	Intra Cranial Pressure
SAS	-	Sub Arachnoid Space
ONH	-	Optic Nerve Head
CRA	-	Central Retinal Artery
CSF	-	Cerebro Spinal Fluid
IOP	-	Intra Ocular Pressure
LGB	-	Lateral Geniculate Body
ATP	-	Adenosine Tri Phosphate
CT	-	Computed Tomography
MRI	-	Magnetic Resonance Imaging
LP	-	Lumbar Puncture
CRV	-	Central Retinal Vein
FFA	-	Fundus Fluoresin Angiography
PAN	-	Poly Arteritis Nodosa
USG	-	Ultra Sono Graphy
VA	-	Visual Acuity
GCA	-	Giant Cell Arteritis
AION	-	Anterior Ischemic Optic Neuropathy
NAION	-	Non Arteritic Anterior Ischemic Optic Neuropathy
RBC	-	Red Blood Cell
VDRL	-	Venereal Disease Research Laboratory
ELISA	-	Enzyme Linked Immuno Sorbent Assay
PNS	-	Para Nasal Sinsuses
TC	-	Total Count
DC	-	Differential Count
NCT	-	Non Contact Tonometer
APH	-	Ante Partum Hemorrhage
PIH	-	Pregnancy Induced Hypertension
PPH	-	Post Partum Hemorrhage

INTRODUCTION

Papilledema is one of the true neuro ophthalmic emergencies[6]. Patients may present to ophthalmologist with signs and symptoms of raised intracranial pressure, such as headache, nausea, vomiting, and abducens paresis, or may be referred by another physician or may be detected accidently during a routine ophthalmic check up. Papilledema serves as an important indicator and warning signal of intracranial pathology. It can also help in finding the severity and management of systemic disease such as hypertension and preeclampsia and preventing further vascular crisis in other organs.

HISTORY

- Albrecht Von Greafe in1860 observed 4 patient of brain tumor and swelling of optic nerve head called "Choked disc" (Stauungs papillae)[1].

- Parson in1908 introduced the term papilledema.

- Paton and Holmes in1911differentiated between Papilledema with increased ICP and optic neuritis. According to them,

Papilledema as a passive edema due to raised intracranial pressure (ICP) without primary inflammatory changes and often without disturbance of function.

Optic neuritis as a swelling of the disc associated with inflammation and loss of function.

DEFINITION OF PAPILLEDEMA

"Papilledema is defined as a passive, non inflammatory edema of the optic nerve head due to raised intracranial pressure, which is almost always bilateral and without visual deficit"[1].

ANATOMY OF OPTIC NERVE HEAD WITH BLOOD SUPPLY

Optic disc is the water-shed zone between retina and optic nerve. It is the exit site of all ganglion cell axons of the retina, which converge at the optic disc to leave the eye and form the optic nerve. The optic disc is located in the nasal retina 3-4 mm from fovea. It is 1.88mm in vertical diameter and 1.76 mm in horizontal diameter. Since there are no photo receptors over the disc, it is projected in visual space as an absolute scotoma, "The Blind spot of mariotte". The blind spot is centered 15^0 from fixation and slightly below the horizontal meridian in the temporal visual field. It represents $7^0/5^0$ in the visual space.

The optic nerves are surrounded by meningeal sheaths, dura, arachonid & pia matter upto lamina cribrosa. There is extension of the Intracranial subarachnoid space (SAS) forward around the optic nerve to

the back of the eye ball. So any increase in ICP compresses thin walled retinal veins, as they cross the extension of SAS leading to congestion and bulging of the disc. Since SA space around optic nerve is continuous with the intracranial subarchonoid space, both eyes will exhibit papilledma. Optic disc unlike the surrounding retina, does not possess the cells of Muller, which holds the nerve fibers together, hence it easily swells in papilledema

Blood supply

Surface area is supplied by retinal capillaries.

Pre laminar region is supplied by peripapillary chorioidal vessels.

Laminar portion of optic nerve head (ONH) receives its blood supply from circle of Zinn, formed by short ciliary vessels.

Post laminar area[8] is supplied by branches of pial plexus from central rential artery (CRA) (Hayresh 1969).

The disc and retina are exposed to intraocular pressure where as the retro laminar and proximal nerve to cerebro spinal fluid pressure (Hayresh and Dass 1960, Hayresh 1963 and 1974).

THEORIES OF PAPILLEDEMA

Earlier theories broadly classified into Non mechanical and Mechanical theories.

Non mechanical theory

1. Gowers theory of inflammation attempted to explain papilledema on the toxic inflammatory basis as caused by toxic elements that were elaborated in the CSF.

2. Jacksons theory of vasomotor instability: He stated the condition to be an upset of nerves controlling the circulation and nutrition of the optic nerve.

These two theories are not supported by any experimental or pathological studies.

Mechanical theory

It has three different hypothesis as follows

1. Those attributing the causal mechanisms essentially to venous obstruction in the region of the optic disc.

2. Those assuming an obstruction of the normal flow of tissue fluid centripetal from the eyes along the optic nerve.

3. Those postulating forcing of cerebrospinal fluid (CSF) into the tissue of optic nerve head to be a part of a general edematous condition of the brain.

Axoplasmic Flow

Normally there is a continuous flow of axoplasm along the axons of optic nerve.

This axoplasmic transport consists of mucopolysaccharides, proteins, glycolipids, gangliosides, phospholipids, cholesterols, smooth endoplasmic reticulum and associated elements.

There are two types of axoplasmic transport

1. **The Anterograde flow or orthograde transport :** Flow from the retinal ganglion cells along the optic nerve axonal fibers to their terminals in the Lateral geniculate body.

   It has two components, slow and rapid.

   Slow components: The slow component materials move at a speed of 1-3 mm per day. It is driven through a peristaltic wave that passively drives across the content and it is the component that is interfered early in the onset of increased intracranial pressure.

Rapid Components: Materials move at a speed of 400mm/day. The velocity of the axoplasmic flow in the fast component depends upon the intra axonal pressure which is determined by difference between the IOP gradient in the pre-laminar area and optic nerve tissue pressure gradient in post laminar area. The optic nerve tissue pressure in turn is determined by CSF pressure in the dural sheaths. The normal tissue pressure in the optic nerve sheath is 6-8 mmHg.

2. **Retrograde flow:** Return flow from the Lateral geniculate body (LGB) to the retinal cells.

HAYREH"S THEORY OF PAPILLEDEMA

- Hayreh has demonstrated the increase intracranial pressure (ICP) in the subarachnoid space(SAS) is easily transmitted to the perineural space. The increased CSF pressure around the optic nerve head causes percolation of the CSF into the optic nerve head, causing increase in the optic nerve tissue pressure.

- This increased optic nerve tissue pressure in the post laminar region causes alteration of pressure gradient across the lamina cribrosa. This causes blockage and stasis of the axoplasmic flow from the retinal ganglion cells to the

Geniculate body. The site of block is in the posterior part of the prelaminar region opposite to the choroid and in the lamina cribrosa (Tso & Hayreh 1977). There is no axonal swelling in the retro laminar optic nerve.

- According to the Hayreh the venous changes are secondary and not primary. According to him as the evolution of papilledema progresses the swollen axons would compress the fine vessels lying in the prelaminar region and in the surface layer causing venous stasis, dilatation of vessels , microaneurysm formation and hemorrhages in the disc and neighboring retina. This leads to formation of fully developed papilledema and extensive hemorrhage, nerve fiber layer infarct and peripapillary edema.

Papilledema occurs only when there is patency of meningeal space surrounding the optic nerve and intracranial structures (Hayreh and Dass 1962 Ernest and Pots 1969). Blockage of this space by adhesion, or tumor prevents papilledema from occurring on side of obstruction.

Papilledema dose not occur in eyes in which antecedent optic atrophy has destroyed most or all of nerve fibers.

To summarise the events leading to papilledema initially following rise of CSF pressure, optic nerve tissue pressure increases leading to stasis of axoplasmic flow in the prelaminar area causing axonal swelling and minimal disc edema. The typical picture of papilledema however, develops later following extra cellular fluid accumulation and ischemia following venous compression.

CIRCULATION OF CEREBROSPINAL FLUID

The craniospinal cavity is almost rigid bony enclosure completely filled by tissue, CSF, and circulating blood. CSF is constantly produced at a rate of 500 ml/day, or 35ml/minute. All the production is by choroid plexus within the lateral ventricles. It is dependent on Na-K-activated ATP ase and Carbonic anhydrase enzyme. CSF flows from the lateral ventricles through the interventricular foramina into the 3rd ventricle and mixes with the CSF produced in that ventricle.

CSF then flows into 4^{th} ventricle through the aqueduct of sylvius out into the subarachnoid space through the foramina of Luschka and Magendie. CSF absorbed passively by the arachnoid granulations that protrude into the venous sinuses and diploic veins, then drain into internal jugular vein and other extracranial veins.

The volume of blood, brain and CSF within the cranial cavity must be in equilibrium.

MEASUREMENTS OF INTRACRANIAL PRESSURE

CSF pressure in adults, measured by lumbar puncture with the patient in the lateral decubitus position. Normal CSF pressure - 80 to 200 mm of water. The CSF pressure may be elevated when the patient

coughs, strains, or holds his or her breath during the procedure. More than 250 mm water is significant.

Normal values have not been well established in children. Less than 200 mm water generally accepted. Irregular variations of CSF pressure were noted in idiopathic intracranial hypertension.

Neuroimaging study, Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) should always be performed prior to LP to make sure of no mass effect in the brain.

LP occasionally induces acquired chiari malformation that may be reversible. Post LP headaches occurs in 40% of patients.

AETIOLOGY OF PAPILLEDEMA

CAUSES OF RAISED INTRACRANIAL PRESSURE[3]

SPACE OCCUPYING LESIONS

- Neoplasm
- Abscess
- Inflammatory mass
- Hemorrhage
- Infarction
- Arteriovenous malformation

FOCAL OR DIFFUSE CEREBRAL EDEMA

- Trauma
- Toxic
- Anoxia

REDUCTION IN THE SIZE OF THE CRANIAL VAULT

- Craniosynostosis
- Thickening of the skull

BLOCKAGE OF CSF FLOW

- Non communicating hydrocephalus

REDUCTION IN CSF RESORPTION

- Communicating hydrocephalus
- Meningeal processes

a. infectious meningitis

(bacterial,fungal,viral and parasitic)

b. inflammatory (aseptic) meningitis

c. carcinomatous meningitis

d. elevated CSF protein

Spinal cord tumours

Guillian Barre syndrome

Chronic Inflammatory Demyelinating

Polyneuropathy(CIDP)

- Elevated venous pressure

INCREASED CSF PRODUCTION

IDIOPATHIC INTRACRANIAL HYPERTENTION

(PSEUDOTUMOR CEREBRI)

INRACRANIAL MASSES[1]

May act as space occupying lesions ,may produce focal or diffuse cerebral edema, and may block the outflow of CSF by direct compression of the CSF drainage pathways or by infiltration of the arachnoid villi or the cerebral venous sinuses. Production of increased protein or blood products secondarily block the arachnoid villi and by directly producing CSF.

Tumors of the brain are the commonest cause, 60% to 80% are associated with papilledema (Gowers,1904; Paton,1909; 77.9% in1,239 cases, Tonnis and Krenkel, 1957) a sign frequently termed **plerocephalic edema[1].**

In Paton"s (1909) series of 252 cases every case of tempero sphenoidal, occipital, cerebellar tumor was associated with papilledema, 87% of frontal tumors, 86% of parietal tumors, 75% of mid brain tumors, 68% of subcortical tumors, 57% of tumors of pons and medulla.

In cerebellar tumors the papilledema develops rapidly. In cerebral tumors it is usually later in appearance and slower in evolution.

Posterior cranial fossa tumor produce an acute papilledema, characterized by an enormous swelling, filiform arteries, engorged veins, plentiful haemorrhages and complicated by Internal hydrocephalus

Craino pharyngeal tumors are often associated with papilledema in contrast to its perhaps unexpected absence in most cases of tumors involving the hypophysis, due to the fact in the former a narrow neck connects the tumor to the stalk of the gland, the bulk of the tumor is ballooned above and acts like a tumor of the third ventricle.

Supra sellar tumor like meningoma, craino pharyngioma, aneurysm can compress optic nerve without evidence of bony changes. To rule out supra sellar mass CT contrast is must. Supra sellar calcification in X-ray seen in 85% of childhood cranio pharyngiomas.

Congenital hydrocephalus rarely associated with papilledema except as a relatively late phenomena after compensation has broken down that its occurrence in an infant suggests the presence of neoplasm (Dandy 1927; Ford and Murphy 1939; Scott 1967; and many others).

Brain abscess is associated with papilledema in a small proportion of cases, 25 to 30% (Kampherstein, 1905). The temporal lobe abscess are most frequently involved. In this condition according to Uhthoff (1914) 80% of cases ipsilaterlly occurs and it has some diagnostic importance.

A solitary tuberculoma usually occurring in cerebellum or pons was found in 4% of cases of papilledema. But Syphilis as represented by

basal gummatous meningitis or a gummatous tumor was more common (Uhthoff, 1914).

Encephalitis and encephalopathies such as Schilder's disease a papilledema may occur (apart from optic neuritis) when the brain becomes oedematus. An aneurysm is an exceptional cause but with an arterio venous aneurysm papilledema occurs more frequently.

Serous meningitis, if any severity frequently produce papilledema small in degree although accompanied by prominent visual symptoms. In this type prognosis is good . The edema subsides after lumbar puncture.

Acute syphilitic meningitis, a rare disease occurring in the early secondary stage or in hereditary infected infants and occasionally appearing as a neuro-Recidive reaction following insufficient treatment. Response to treatment is good.

Tuberculous meningitis is a rare cause (23%of cases according to Blagojevic and Armbasic, 1956), papillitis being the commoner complication.

Infective traumatic meningitis can produce papillitis and papilledema

Head injury[1]

Cerebral haemorrhage can give rise to papilledema, is frequent ,if the blood enters the optic nerve sheaths. Papilledema develops within 48 hours after injury, the cause is usually an extra or intra cerebral haemorrhage and immediate surgical measures are indicated.

If signs of pressure appear towards the end of first week, they are evidence of traumatic cerebral edema at this stage there is no indication for operation.

If they appear in the second week they are usually indicative of increase in the production of cerebro spinal fluid and often the sign of infection.

If they appear in the third week they are usually accompanied by the formation of a cerebral abscess.

In thrombosis of cavernous sinus papilledema is relatively rare as it also in thrombosis of the dural venous sinuses (Ford and Murphy).

In subdural bleeding after injuries to head, it is probably more common than is usually supposed owing to the absence of routine ophthalmic examination. Furlow found mild papilledema in 68% of cases. An disc edema can occur with a sub arachnoid haemorrhage.

Parasitic infestations are a rare aetiolgic factor. Cysticercosis found majoritly in the 4thventricle, hydatid cyst also found in ventricles gave rise to papilledema.

Crypotococcosis (torulosis) can result in meningitis with papilledma, neuroretinitis and optic atrophy with marked impairment of vision

Changes in the cerebro- spinal fluid resulting in papilledema are seen in the Guillian- Barre syndrome (1916). A form of polyradiculitis where in the protein content of the CSF rises, producing increased viscosity and defective absorption or edema of the brain substance.

Spinal cord tumor Cervical or more caudally cauda equina tumor occasionally give rise to papilledema.

CEREBRAL VENOUS THROMBOSIS

Primary hematological

Anti phospholipid antibody syndrome, Thrombophilia, Thrombocythemia, Polycythemia, Disseminated intravascular coagulation, Hyperviscosity syndrome.

Systemic conditions associated with coagulopathy

Behcet’s disease, Systemic lupus erythematosus, Neurosarcoidosis, Cancer, Pregnancy/postpartum, Nephrotic syndrome, Infections, Post surgery.

Local infections

Mastoiditis, Facial/orbital cellulitis, Meningitis.

Traumatic

Tumours (compression of a sinus)

Dural arteriovenous fistula

Transverse sinus stenosis

Occlusion of internal jugular vein

Iatrogenic, Indwelling catheter, Surgery, Traumatic, Tumors (extravascular).

Increased venous pressure

Right cardiac insufficiency, Superior vena cava syndrome, Morbid obesity, Hyper viscosity syndrome.

MR venogram of dural venous sinuses is must

IDIOPATHIC INTACRANIAL HYPERTENSION / PSEUDOTUMOR CEREBRI

Definition

A syndrome in which patient present with symptoms and signs of elevated intracranial pressure, the nature of which may be either idiopathic or due to various causative factors.

Pseudo tumor cerebri is diagnosed by following criteria

- Normal head imaging scan
- Increased intracranial tension as measured on lumbar puncture[17]
- Normal cerebro spinal fluid composition
- No evidence of intracranial mass lesion or cerebral venous thrombosis

Symptoms

Severe headache, diplopia, transient episodes of visual loss, pulsatile tinnitus, nausea and vomiting. Occurs predominantly in obese women19.

Signs

Papilledema due to raised intracranial pressure Unilateral or bilateral sixth nerve palsy may be present

Associated factors

Obesity, significant weight gain, and pregnancy often are associated with idopathic form.

Possible causative factors include

Hormonal abnormalities

Impaired absorption of cerebro spinal fluid

Chronic obstructive pulmonary disease

Radical neck dissection

Cortico steroid withdrawal[5]

Use of medications such as vitamin A, tetracycline, nalidixic acid, cyclosporine and oral contraceptives.

Foster Kennedy syndrome

Tumor usually a frontal lobe glioma, sphenoidal or olfactory groove meningioma compresses the optic nerve causing atrophy on the side of tumor. First it occurs before the tumor takes up a significant amount of intracranial space then as the tumor grows, the ICP increases and develops papilledema on contralateral side while atrophic side does not.

PATHOLOGY

The lamina cribrosa separates the optic nerve into its intraocular and retroocular portions. The tissue pressure within the intraocular portion of optic nerve is higher than that posterior to the lamina. This normal pressure gradient at the level of lamina reflects the influence of the intraocular pressure, which exceeds the CSF pressure.

Lowering the tissue pressure in the prelaminar area or elevating the pressure n the retrolaminar area of the optic nerve disrupts axoplasmic flow gradient and results in disc swelling. Edema of the disc may occur when prelaminar tension is abruptly elevated as in acute glaucoma.

Passive swelling of optic disc may be classified as Prelaminar and Retrolaminar causes

UNILATERAL PAPILLEDEMA (Pre laminar caused by intraocular disease)	**BILATERAL PAPILLEDEMA** (Retrolaminar caused by intra orbital, intracranial, spinal cord disease)
A. Hypotony External fistula,trauma, uveitis, Retinal detachment **B. Acute glaucoma** **C. Local and systemic vascular disease** Malignant hypertension Venous stasis and Hemorrhagic retinopathy (occlusion of CRV) Anterior ischemic optic neuropathy Collagen disease Sarcoid, Giant cell arteritis, Lupus erythematosus, PAN. Hematological disease Anemia, Polycythemia vera, Leukemia, Macroglobulinemia Cardio pulmonary disease Emphysema, Congenital heart defects, cystic fibrosis	**A. Tumor or Pseudotumor or Endocrine ophthalmopathy** Increased ICP secondary to SOL Inflammation-meningitis,encephalitis, abscess Incrased viscosity of the CSF Trauma with meningeal or intracerebral haemorrhage Vascular lesion or malformation or aneurysm **B. Internal hydrocephalus (Idiopathic)** Secondary to decreased volume of intracranial space Craniostenosis , Oxycephaly , Crouzons' desease , Paget's disease **C. Pseudotumor cerebri**

Histological section of optic nerve with papilledema shows

- Papilledema is the simple edema of the optic nerve head with an edematous swelling of the nerve fibers and an infiltration of all tissues with fluid.
- The physiological cup is smaller or obliterated. The tissues of the disc project into the cavity of the eye, displacing retina laterally and throwing it up into small folds.
- The axial fibers becomes raised to fill the central depression and the peripheral fibers, instead of forming a regular arc between the retina and the nerve, form a double S shaped curve, bending laterally and curving acutely backwards before they pass around the termination of Bruch"s membrane to meet the nerve.
- Compression, detachment and lateral displacement of the peripapillary retina[2] appear to be major reasons that blind spot increase in size in patient with papilledema.
- However the blind spot may be enlarged even there is no obvious retinal displacement or datachment. In this setting, the enlarged blind spot represents a refractive scotoma caused by acquired peripapillary hyperopia from elevation of retina by peripapillary

sub retinal fluid. If you use progressively stronger plus lenses the blind spot can be reduced to near normal size.

- Both veins and capillaries are distended, the haemorrhage may overlie and obscure the optic disc and may occur in all layers of peripapillary retina. Occasionally the haemorrhage extending into the vitreous or adjacent sub retinal space.

- Focal necrosis of nerve fiber is frequent. Lateral out pouching of nerve substance adjacent to the retina. Small vessels on optic disc often appear prominent with abnomally plump and proliferative endothelial cells (Cogan and Kuwabara, 1977).

- The substance of prelaminar portion of optic nerve is clearly swollen, but does not show any increase in cellularity until secondary gliosis occurs.

- Post laminar optic nerve does not participate in the swelling and usually appears normal unless secondary optic atrophy has occurred.

- Vaginal space about the nerve is often distentded with the stretching of delicate arachnoid strands that bridge the subarachnoid space.

- Electron microscopy studies indicate that most of the increase in tissue elevation that occurs in papilledema results from intra axonal swelling and not from extra cellular edema.

- Swelling marked in superficial optic nerve head, some of the axons increase to 10-20 times their normal diameter. The intra axonal swelling is accompanied by increase in mitochondria, disorganization of neuro filaments and the accumulation of dense intracellular membrane- enclosed bodies.

- In severe papilledema cystiod bodies occurs in those portion of optic disc that bulge laterally towards adjacent retina. These cystoid bodies and resultant necroses appears to result from strangulation of the angulated nerve fiber.

- The disc edema cannot occur in atropic optic nerve head. High myopic eye, senile eye, disc edema less pronounced because of the changed condition of those tissue.

CLINICAL FEATURES OF PAPILLEDEMA

SYMPTOMS AND SIGNS OF PAPILLEDEMA

Non visual manifestations

- Headache -Earliest symptom of increased ICP. It is caused by stretching of meanings. Headache increased by coughing, straining, and valsalva maneuvers in some patients.
- Nausea and vomiting- associated with increased ICP. Projectile vomiting is rare[3]. Vomiting, bradycardia, difficulty swallowing, and respiratory distress occurs by herniation of medulla into foramen magnum.
- Loss of consciousness, generalized motor rigidity and pupillary dilatation
- Loss of consciousness-compression of cerebral cortex and reduction of blood supply.
- Generalized motor rigidity- Herniation of hippocampal gyrus through the tentorium, tentorial herniation places pressure on crura cerebri.
- Pupillary dilation- Direct pressure on the oculo motor nerves or dorsal midbrain produces bilaterally dilated pupils.

- CSF rhinorrhea- Trauma, Congenital anomaly at the base of the skull.

VISUAL MANIFESTATIONS

- Asymptomatic in early and even fully developed papilledema.
- Brief, transient obscurations of vision
- Positive visual phenomena- photopsias, phosphenes, and scintillating scotoma.
- Visual field defect
- Diplopia- compression or stretching of the abducens nerve at the base of the skull. Damage may be unilateral or bilateral.
- Trochlear nerve palsies may rarely occur-compression of either dorsal midbrain or nerve themselves by a ballooned suprapineal recess.

OPHTHALMOSCOPIC SINGNS OF PAPILLEDEMA[4]

Mechanical signs[4]	Vascular signs
1. Elevation of disc and mushrooming of the nerve head into vitreous.(3D =1mm)	1. Reddish discolouration (Hyperemia) of the optic disc.(Capillary stasis)
2. Blurring of optic disc margin.	2. Venous (congestion), Dilation and toruosity increased in caliber of veins and arteries
3. Filling in of the physiological cup.	3. Peripapillary haemorrhages (margin of disc) and within the disc.
4. Edema of the peripapillary Nerve fiber layer	4. White exudates in the disc / on peripapillary area.
5. Retinal and /choroidal folds	5. Nerve fiber layer Infarction

Phenomenon of parallax

Difference in the plane of retina and of the never head in which the retinal vessels coarse leads to the phenomenon of the parallax. 3 dioptre corresponds to1 mm of prominence. Ophthalmoscope is first focused on the most prominent vessels on the optic nerve head, and the strongest plus or weakest minus lens giving a clear picture of this vessels is noted. We must wear spectacle correction glass, select a vessel parallel to the first observed, near the disc margin in the retinal plane and again to find the

strongest plus or weakest minus lens allowing a clear image of vessel. The difference in strength of these two lenses expresses the prominence of disc in dioptre. It can be converted to millimeter.

Reddish discoloration of disc (plethora of the disc is important early sign). Normal ratio of veins to arteries is 3:2. It is increased to 4:2 or even 5:2 increased toruosity. Deflection of vessels caused by prominence. Haemorrhage may be present at or near the disc in rapidly developing chocked disc. It may be an important early sign.

Soft white patches seen on disc or at its margin in later stages are definitely not exudates. They consist of varicosities called cystoid bodies resulting from terminal nerve fibers swelling of Cajal identified with those of cotton wool exudates in the retina.

White or grayish white spots in retina surrounding the disc are remnant of absorbed haemorrhage. If edema increases it extends to adjacent retina to reach macular area . Milky appearance with striate pattern due to arrangement of nerve fiber. Droplets of fluid accumulate underneath the ILM between disc and macula visible as minute brilliant white dots in a radial arrangement and assume the shape of a fan, resolve

completely with resolution of papilledema. This picture contrast with macular fan seen in malignant hypertension which is composed of lipids and lipid laden macrophages.

Classical traid of perimetric changes in disc edema

- Enlargement of blind spot.
- Gradual slopes of the isopters around the blind spot.
- Defects in spatial summation (photometric disharmony).

Macular edema produce metamarphopsia and a relative central scotoma with slight diminished central vision. Advanced stage of papilledema the enlargement of blind spot and central scotoma may merge to form a relative ceco central scotoma.

STAGES OF PAPILLEDEMA[9] (according to duration of papilledema)

Early papilledema

- Visual symptoms absent and visual acuity normal
- Optic disc show hyperemia and mild elevation[11]

- Disc margins (initially nasal, later superior, inferior and temporal) appear indistinct
- Loss of spontaneous venous pulsation (20% of normal population do not have venous pulsations, preservation of venous pulsation rules out papilledma).

Established papilledema

- Transient visual obscurations may occur in one or both eyes, lasting a few seconds, often on standing or bending forwards.
- VA normal or reduced
- Optic disc shows severe hyperemia, moderate elevation with indistinct margins and obscuration of small surface vessels.
- Venous engorgement, peripapillary flame shaped haemorrhages and frequently Cotton wool spots.
- Optic nerve head enlarged and peripapillary retina exhibits radial concentric folds **(paton's lines)**.
- Hard exudates may radiate from the center of fovea in the form of a **"macular fan"**[34]**-** an incomplete star with temporal part missing.
- The blind spot is enlarged.

Chronic papilledema

- VA is variable and visual fields begin to constrict.
- Optic discs are markedly elevated with a champagne cork appearance.
- Cotton wool spots and haemorrhages absent
- Opto ciliary shunts and drusen like crystalline deposits (corpora amlyacea) may be present on the disc surface.

Atrophic (secondary optic atrophy)

- VA severely impaired.
- The optic discs are dirty grey colour, slightly elevated, with few crossing blood vessels and indistinct margins.

DIAGNOSIS OF PAPILLEDEMA

- History and physical examination ,BP measurement
- Ocular examination pupillary and color vision assessment, posterior vitreous evaluation for cells.
- Careful opthalmoscopic examination clinically (includes red free ophthalmoscopy and slit lamp biomicroscopy with handheld lens and Indirect ophthalmoscopy).
- Early papilledma by fluorescein angiography.

According to most investigators earliest frames of FFA shows

Disc capillary dilation,

Dye leakage,

Microaneurysm formation.

Late frames show leakage of dye beyond the disc margins. Hayreh 1976 observed FA did not show any abnormalities until optic disc swelling was of a “mild to moderate degree”.

- Orbital echography – show increased diameter of optic nerve, also detect buried optic disc drusen.

- Confocal scanning laser tomography (CSLT; Hiedelberg retinal tomography) and Optical Coherence Tomography[27] –useful in diagnosis of disc edema.

- Emergency MRI with gadolinium and magnetic resonance venography (MRV) of the head are preferred. CT scan may be done if MRI not available[12].

- LP with CSF analysis and opening pressure measurement if the CT or MRI /MRV do not reveal a mass lesion or hydrocephalus.

DIFFERENTIAL DIAGNOSIS OF PAPILLEDEMA

1. Pseudopapilledema-(Optic disc drusen[7], Congenitally anomalous disc) : Not true disc swelling, vessels overlying disc not obscured, disc not hyperemic, spontaneous venous pulsations often present. Buried drusen identified with B scan USG[18].

Hypermetropic eyes: Appearance of swelling and blurred margins largely due to reflux. Swelling never >2D, no venous engorgement, edema, or exudates, blind spot is normal, FFA –no leakage.

2. **Papillitis[10]:** afferent pupillary defect, decreased color vision, decreased VA , unilateral.

3. **Hypertensive optic neuropathy:** Extremely high blood pressure, narrowed arterioles, arterio venous crossing changes, hemorrhages with or without cotton wool spots extending to peripheral retina.

4. **Central retinal vein occlusion:** Hemorrhages extent far beyond the peripapillary area, dilated tortuous veins, unilateral, with acute loss of vision.

5. **Ischemic optic neuropathy:** Disc swelling pale, hyperemic, initially unilateral unless due to GCA, with sudden visual loss.

6. Leber optic neuropathy: Young men, initially unilateral but rapidly bilateral, rapid and progressive visual loss, disc swelling associated with peripapillary telangiectasias.

7. Diabetic papillopathy: The most commonly proposed theory suggests diabetic papillopathy to be a mild form of non arteritic AION with reversible ischemia of both prelaminar and inner surface layers of optic nerve head. Edema of optic nerve head[20] in the absence of significant visual dysfunction. Prominent surface telangiectasias may represents vascular shunting from pre laminar to ischemic vascular beds. Crowded optic disc in the fellow eyes as in NAION support ischemic mechanism. Unilateral or bilateral disc edema in young type I diabetics, without usual defect in visual field and pupillary function. It is associated with non AION or optic neuritis. Older patient with type II diabetes mellitus also included.

Criteria for diagnosis of diabetic papillopathy

Presence of diabetis mellitus 70% in type I, 30 % in type II

Optic disc edema (unilateral in 60%)

Only mild optic nerve dysfunction.

8. Thyroid related optic neuropathy: Eye lid retraction, ocular misalignment, resistance to retropulsion.

9. Amiodarone toxicity: sub acute visual loss, and disc edema.

TREATMENT

Papilledema: Treatment : According to the cause medical and surgical.

Pseudo tumor cerebri

Weight reduction - if overweight

Medical

Antiedema measures like IV Mannnitol, Tab. Furosemide(40mg), injection Decadran (8mg), Tab. Acetazolamide(250mg).

Surgical[16]

Ventriculo peritoneal shunt in case of intractable headache, optic nerve sheath decompression if vision is threatened.

Major complications of papilledema are blindness due to secondary post papilledemic optic atrophy and 6th nerve palsy.

Role of Ophthalmologist

Monitor all parameters of optic nerve function

Visual acuity

Colour vision

Quantitative perimetry for field assessment

Fundus photograph to document the changes in disc.

Important to know the Pseudotumour cerebri is a diagnosis of exclusion. All patients with papilledema should be considered to have an intracranial mass until proven otherwise.

AIMS OF THE STUDY

1. To study the aetiological pattern in Papilledema
2. To study the involvement of 6th cranial Nerve in Papilledema
3. To study the visual acuity pattern in papilledema
4. To study the visual field and colour vision pattern in papilledema

MATERIALS AND METHODS

The cases studied were the patients with Papilledema who presented to department of neuro ophthalmology at the Regional Institute of Ophthalmology and Govt. Ophthalmic Hospital. All the age groups and both sexes were included. A complete ophthalmological workup was done.

INCLUSION CRITERIA

1. All cases of papilledema.

EXCLUSION CRITERIA

All cases of post papilledemic optic atrophy, papilledema due to grade IV hypertensive retinopathy, and papilledema due to grade IV hypertensive retinopathy in pregnancy induced hypertensive patients were excluded.

REGISTRATION

Name

Age

Sex

Occupation

Address

HISTORY OF PRESENT ILLNESS

The common complaints were:

a. Headache- location, nature, any radiation, aggravating and relieving factors, is it increasing while coughing , straining, and Valsalva maneuver, and association with nausea/vomiting.

b. Double vision-whether uniocular / binocular, constant/intermittent, fluctuating or not, more for near or distance, whether images were horizontally or vertically separated, where it is increased on any particular direction, onset and progress.

c. Defective vision – apart from double vision, any blurring of vision.

d. Deviation of eyeball-right/left eye, duration

e. Transient visual obscuration/visual loss in seconds.

f. Loss of consciousness / flashes of light and any visual field defect.

g. Positive phenomenon like photopsia

Details of the progress from onset, the treatment undergone to the present state is noted. Any other significant medical/surgical history is also recorded.

PAST HISTORY

H/o diabetes, hypertension, tuberculosis, syphilis, AIDS, malignancy in the present or past.

H/o migraine or neurologic disease

H/o exanthematous fever and endocrine disorders

Birth history – trauma/ hydrocephalus/normal or forceps delivery.

PERSONAL HISTORY

Diabetes, smoking, alcoholism etc.

Exposure to pet animals /non vegetarian.

In female- obstetric history- menstrual cycle, PIH, PPH, APH.

GENERAL EXAMINATION

General vital data like pulse, blood pressure, peripheral pulses are noted. Higher function status also noted.

OCULAR EXAMINATION

- Head posture, facial symmetry are noted.
- Any deviation of eyeball is recorded. Under slit lamp, details of the anterior segment from the lids to the lens are noted.
- Extraocular movements are noted down-both ductions and versions in all cardinal positions.
- Pupil size, reaction, any anisocoria is noted.
- A dilated fundus examination and refraction is done.
- Diplopia charting – is done in a dark room. Patient is asked to wear goggles with red in front of the right eye and green before the left eye.

  A torch light with a staenopic slit is used. The patient is asked to look at this torch held 120 cm away and then the torch is moved to various positions. The patient is asked to describe the position of the images. The false image is usually the fainter and farther one.

- If 6 th C Nerve palsy is suspected restriction of abduction is noted.
- If a superior oblique palsy is suspected, Parks Bielchowsky's 3 step head tilt test is done.
- A forced duction test is performed in doubtful cases to rule out restrictive etiology.

NEUROLOGIC EXAMINATION

Examination of other cranial nerves,

Motor, sensory, cerebellar symptoms and signs are noted.

EXAMINATION OF THYROID- Any neck swelling is noted.

Examination of spine & back is noted.

To look for congenital anomalies and neurocutanous markers.

Examination of ENT structure was done.

INVESTIGATIONS

Evaluate Both eyes for all cases.

1. Vision - a. Uncorrected (Using Snellen's charts at 6 metres)

   b. Best corrected (after Retinoscopy) was done.

2. Intraocular pressure was measured with applanattion tonometer after topical anaesthesia

3. Detailed slit lamp examination

   Lid

Conjunctiva

Cornea

Iris

Pupil

Anterior Chamber

Lens/Pseudophakia/Aphakia were noted.

4. Fundus examination- papilledema, any abnormalities were noted

5. Diplopia charting was done .

7. Measurement of deviation-primary & secondary deviation, cover uncover test in various gaze positions, for near and distance as well.

8. Hess charting

9. Gonioscopy

10. Visual field examination

11. Colour vision

BLOOD TEST : (For all cases)

Total count

Differential count

Erythrocyle sedimentation rate

Haemoglobin %

RBC count / platelet count

Blood sugar – Fasting, Postprandial.

Mantoux intradermal test

Blood VDRL / ELISA

CSF analysis (if any)

Urine – albumin/ sugar, Motion- ova/ cyst

RADIOLOGY

X ray skull lateral view / orbit

X ray chest – tuberculosis

X ray PNS – (paranasal sinuses) – mucococle, antral growth, sinusitis, orbital floor fractures.

ORBITAL USG – (in indicated cases)

NEURO IMAGING:

CT

MRI

MRA/MRV

Fundus Fluorescin Angiography

Specialist opinion

Neurophysician/Neurosurgeon

Radiologist

FOLLOW UP

Follow up was done with the following parameters.

Recording of patient's complaints-whether stable/improving or worsening

Vision

Pupil assessment

Extraocular movements

Diplopia charting

Fundus

Visual field testing

Colour vision

RESULTS

A prospective study of 45 cases of papilledema were examined. The following results were obtained.

1. AGE DISTRIBUTION

The following table shows the age distribution in papilledema patients.

TABLE – I

Age group	Total cases
0-10	2
11-20	9
21-30	20
31-40	9
41-50	4
51-60	1
	45

Regarding the age distribution, considering all the papiledema cases in total, the maximum number of patients belonged to 21-30 years age group (44.4%) followed in frequency by 11-20 and 31-40 years age group both with 20% of patients, 41-50 years age group with 8.9% patients and 0-10 years age group with 4.4% patients. The least number was seen in the age group of 51-60 years (2.2%).

2. SEX DISTRIBUTION

The following table shows sex distribution in papilledema patients.

TABLE - II

MALE	FEMALE	TOTAL
15	30	45

In the study there was a gender difference, out of 45 patients 30 patients (66.7%) were in the female group. Out of 45 patients 15 patients (33.3%) were in the male group. Male to female ratio was 1:2.

3. LATERALITY

The following table shows laterality in papilledema patients.

TABLE - III

Unilateral papilledema	Bilateral papilledema	Total
2	43	45

Regarding laterality bilateral involvement was most common, 43 patients out of 45 patients had bilateral papilledema (95.6%). Unilateral papilledema was found in 2 cases out of 45 cases (4.4%) (Foster Kenndy syndrome- ipsilateral optic atrophy and contra lateral papilledema).

4. AETIOLOGICAL PATTERN IN PAPILLEDEMA

The following table shows aetiological pattern in papilledema patients.

TABLE – IV

Aetiology	**No.of cases**
Space occupying lesion(SOL)	13
Meningitis(MTIS)	6
Thrombosis(TSIS)	4
Trauma(TMA)	2
Post surgical(PS)	2
IICT	7
Systemic condition(SC)	1
CT Normal(CTN)	7
Not turn off (NTO)	3
	45

In the study we observed 13 cases (28.9%) of papilledema to have space occupying lesion. 6 cases (13.3%) of papilledema to have meningitis. 4 cases (8.9%) of papilledema to have thrombosis. 2 cases (4.4%) of papilledema to have trauma. 2 cases (4.4%) of papilledema to have post surgical causes. 7 cases (15.5%) of papilledema to have idiopathic intracranial hypertension. 1 case (2.2%) of papilledema to have systemic condition- leukemia. 7 cases (15.5%) of papilledema to have normal CT brain. 3 cases (6.7%) of papilledema were not turn off.

5. SIXTH CRANIAL NERVE PALSY IN PAPILLEDEMA

The following table shows sixth cranial nerve palsy in papilledema patients.

TABLE – V

Sixth CN palsy	Sixth CN normal	Total
20	25	45

In this study 20 cases (44.4%) of papilledema had sixth cranial nerve palsy. VI cranial nerve was normal in 25 cases (55.6%) of papilledema.

6. VISUAL ACUITY IN PAPILLEDEMA

The following table shows visual acuity pattern in papilledema patients.

TABLE - VI

Vn 6/6	Correction 6/6	NIG	Total
24	14	7	45

In the study 24 cases (53.3%) of papilledema to have normal visual acuity 6/6. 14 cases (31.1%) of papilledema to have 6/6 visual acuity with correction. 7 cases (15.6%) of papilledema to have not improving with glasses.

7. VISUAL FIELDS IN PAPILLEDEMA

The following table shows visual fields pattern in papilledema patients.

TABLE - VII

WNL	B/S Enlarged	Defective	Not co-op	Total
20	13	6	6	45

In the study the visual field pattern was assessed, 20 cases (44.4%) of papilledema had normal visual field pattern. 13 cases (28.9%) of papilledema had only blind spot enlargement. 6 cases (13.3%) of papilledema had defective visual field pattern. 6 cases (13.3%) of papilledema to have not cooperative for visual field testing.

All cases of pailledema had normal colour vision assessed by using ischihara charts.

DISCUSSION

1. AGE

In this study 45 cases of papilledema were examined. The majority of patients belonged to 21-30 years of age group. But usually in case of idiopathic intracranial hypertension the most common age group affected was 30 years.

2. SEX

In this study there was gender difference with female preponderance 66.7% females compared to 33.3% males. In our series male to female ratio 1:2. Usually in idiopathic intracranial hypertension females are more commonly affected.

3. LATERALITY

In this study bilateral papilledema was most commonly (95.6%) seen, where as unilateral papilledema (Foster Kennedy syndrome) was least commonly (4.4%) seen.

4. AETIOLOGICAL PATTERN IN PAPILLEDEMA

- In this study 28.9% of papilledema belonged to space occupying lesion. Comparing with the study by Gowers, 1904; Paton,1909; 77.9% in 1,239 cases of papilledema belonged to tumours of brain.

- 13.3% of papilledema were due to meningitis. Comparing with the study by Blagojevic and Armbasic, 1956, they found 23% of cases belonged to tuberculous meningitis. In this study out of 6 patients, 5 patients had TB meningitis, 1 patient had HIV positive with disseminated TB with cryptococcal meningitis.

- 8.9% of papilledema belonged to thrombosis. Out of 4 patients, 3 patients belonged to cerebral venous sinous thrombosis[14], one patient belonged to anti phospholipid antibody IgM positive.

- 4.4% of papilledema belonged to trauma. Out of 2 patients one patient had post traumatic bilateral SDH, one patient had post traumatic bilateral frontal lobe contusion.

- 4.4% of papilledema belonged to post surgical causes. Out of 2 patients one patient developed papilledema after modified radical neck dissection for papillary carcinoma thyroid, another patient developed papilledema after decompression for arnald chiary malformation[13].

- 15.5% of papilledema belonged to idiopathic intracranial hypertension. Out of 7 patients one patient had papilledema due to Vit A over dosage. One patient had papilledema with empty sella.

CT and MRI/MRV was normal for remaining 5 patients. LP was done in one patient showed the opening pressure 32 cm H2O.

- 2.2% of papilledema belonged to systemic condition. In this study one patient developed papilledema due to acute myloblastic leukemia.

- 15.5% of papilledema belonged to normal CT brain group. In this group MRI was not done. According to Rev Prat 2001 Dec 15 ; 51 (20) : 2210-4 isolated bilateral papilledema require MRI, looking for tumour, hydrocephalus, cerebral venous thrombosis, if MRI is normal and does not show any tonsilar herniation, lumbar puncture has to be done with CSF pressure evaluation. Intracranial hypertension with out any intracranial lesion (mass lesion, ateriovenous shunt, venous thrombosis) is pseudotumour cerebri syndrome[15].

- 3 cases (6.7%) of papilledema were not turn off.

5. SIXTH CRANIAL NERVE PALSY IN PAPILLEDEMA

In this study involvement of sixth cranial nerve palsy was assessed clinically by abduction restriction and diplopia charting. 44.4% of papilledema had sixth cranial nerve palsy. VI cranial nerve was normal in 55.6% of papilledema.

Damage may be unilateral or bilateral, the mechanism being compression or stretching of the abducens nerve at the base of the skull.

6. VISUAL ACUITY IN PAPILLEDEMA

In this study the visual acuity was tested by using snellen's chart at 6 meter distance. 53.3% of papilledema belonged to normal visual acuity 6/6. 31.1% of papilledema belonged to 6/6 visual acuity with correction. 15.6% of papilledema belonged to visual acuity was not improving with glasses. Normally patients with early and even fully developed papilledema are visually asymptomatic, the visual acuity not being affected. In this study 7 patients visual acuity were not improving with glasses. Out of 7 patients 3 patients visual acuity were not improving due to macular edema. 2 patients visual acuity were not improving due to cataract. 2 patients visual acuity were not improving due to pallor of disc on the affected side of SOL (Foster Kennedy syndrome), fellow eye vision was improving to 6/6.

7. VISUAL FIELDS IN PAPILLEDEMA

- In this study the visual field pattern was assessed by automated perimetry and jerrums screen. 44.4% of papilledema showed normal visual field pattern. 28.9% of papilledema showed blind spot enlargement. 13.3% of papilledema showed defective visual field pattern. Out of 6 patients 2 patients showed typical of

contracted visual field defect, 3 patients showed defective field and one patient had homonymous hemianopia with papilledema due to empty sella. 13.3% of papilledema were not cooperative for visual field testing.

- Normally in early stage of papilledema – there is no field defect. In established stage, there is enlargement of blind spot. In chronic stage, there is associated with peripheral constriction of the visual field with appearance of nerve fiber layer bundle defects. Finally, there is total loss of visual field.

In this study all patients of papilledema showed normal colour vision pattern.

CONCLUSION

1. Papilledema occur in a wide range of age but are more common in the age group of 21-30 years.
2. Overall, females were affected more than males.
3. Bilateral papilledema was most common than unilateral papilledema like Foster Kennedy syndrome.
4. The common aetiological factor for papilledema was space occupying lesion.
5. 44.4% of the patients had sixth cranial nerve paresis in papilledema.
6. More than three forth of patients had normal visual acuity in papilledema.
7. Less than half of the patients had visual field involvement in papilledema.
8. All the patients had normal colour vision pattern in papilledema.
9. A careful history, general and complete ophthalmological workup with necessary investigations like CT, MRI/MRV are mandatory to diagnose patients with papilledema. Since papilledema can be manifestations of life threatening condition, the ophthalmologist should be able to detect early papilledema and refer them immediately.

BIBLIOGRAPHY

1. System of ophthalmology by Sir Stewart Duke- Elder, Vol. XII – Neuro Ophthalmology, Page 32- 62.

2. Walsh & Hoyt's clinical Neuro Ophthalmology Vol. One, Page 487- 538.

3. Text Book of Ophthalmology, Vol. One ; Sunita Agarwal, Athiya Agarwal; Page 360- 363.

4. Walsh & Hoyt's clinical Neuro Ophthalmology The Essentials, Second Edition Page 122- 145.

5. Liu- Volpe- Galetta – Neuro Ophthalmology Diagnosis & Management, Page 206.

6. Neuro Ophthalmology by Desmond P- Kidd, Nancy J. Newman, Vallevie Biousse; Page 280.

7. Albert Jackobiec's Principle & Practice of Ophthalmology, Vol. III, Third Edition, Page 3866- 3868.

8. Wolff's Anatomy of the Eye & Orbit, Eighth Edition by Antony J. Brow, Page 528

9. Clinical Ophthalmology Sixth Edition, Jack J. Kanski, Page 799- 802.

10. Parson's Diseases of the Eye, Twentieth Edition, by Ramanjit Sihota Page 329- 335.

11. American Academy of Ophthalmology Section- 5, 2005- 2006, Neuro Ophthalmology, Page 111- 120.

12. The Wills Eye Manual, Fifth Edition, Jastia P. Ehlers & Chirag P. Shah, Page 252- 256.

13. Am. J.Oph. 2007 May 133 (5) Page 673- 8, Resolution of papilledema after Neurological Decompression for Primary Chari Malformation.

14. J. Neurology 2008, Transverse Venous sinus stenosis and increased intracranial hypertension which comes first.

15. Rev. Prat 2001 Dec. 15; 51(20): 2210-4. Regarding Pseudotumour Cerebri Syndrome.

16. Curr. Neuro Neurosis Rep: 2004 Mar; 4 (2):111- 6. Pseudotumour Cerebri.

17. Curr Opin Ophthalmol 1998 Dec; 9 (6): 27- 32, Elevated intracranial pressure & Pseudotumour cerebri.

18. Clinical Eye & Vision Care Vol. 11 issue 2, 20 August 1999; page 109- 113, Optic Nerve head drusen causing disc Hemorrhage, disc edema.

19. C. Bulens, W A De Vries, H Van Crestel ; Benign intracranial Hypertension, A Retrospective & follow up study.

20. Diabetic Papillopathy, RE appen, SR Chandra, R Kelein, FL Myers, Albrecht Von Craetes Arch Klin Exp. Ophthalmol, 1978 Mar 7; 205 (4): 263- 77.

21. Neurology India. Yr 2002 Vol. 50, issue 4 page 528- 9, Visual loss with papilledema in Guillain Barre Syndrome.

22. Am J 2003 Apr; 135 (4): 437- 46, Anemia & Papilledema.

23. Acta Neurologica Scandinavia Vol. 50 issue 5, page 642- 650, Vit-A intoxication causing papilledema & stimulating acute encephalitis.

24. Hand Book of Ocular disease management, optic disc edema & papilledema.

25. Neuro ophthalmology 1983 Vol. 3, No: 3, page 199- 204, a case of Dural venous malformation with papilledema.

26. Headache ISSN 0017- 8748 2009, Vol. 49, $n^0$2, pp- 185- 193 a comparison of idiopathic intracranial hypertension with and without papilledema.

27. Invest ophthalmol Vis . 2001 Jun; 42(7): 1487- 94 Correlation of optic nerve Tomography with visual field sensitivity in papilledema.

29. Arch ophthalmol 2001 Jan; 129(1): 59-63 Acute idiopathic blind spot enlargement syndrome: a review of 27 new cases.

30. I J O Year 2008, Vol. 56, issue 4: page 327- 329 Polycythemia vera presenting with bilateral papilledema a rare case report.

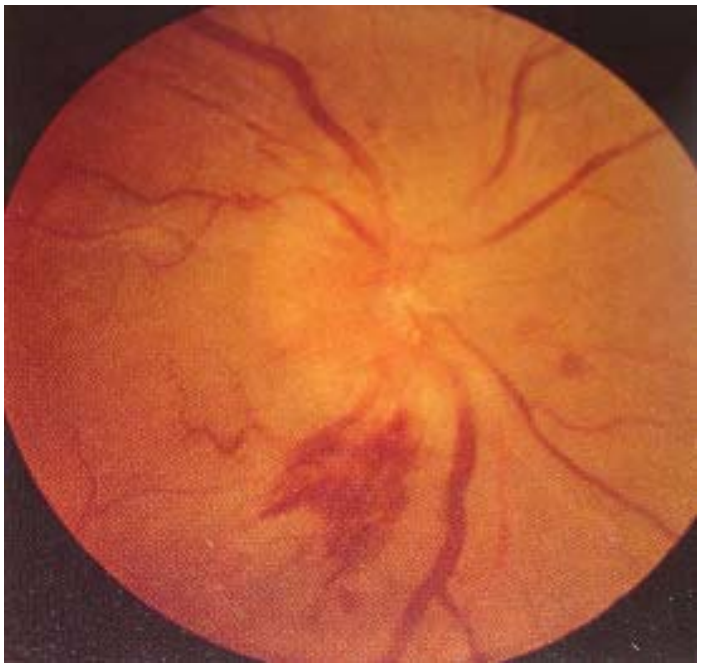
31. Journal of Neurosurgery November 2007 Vol. 23, No 5, Mechanism of visual loss in papilledema.

32. B JO 201: 10, 1136/ 2008, 140053, Papilledema with vision loss with elevated cerbrospinal fluid protein in a patient with SLE. Diagnosis and management challange.

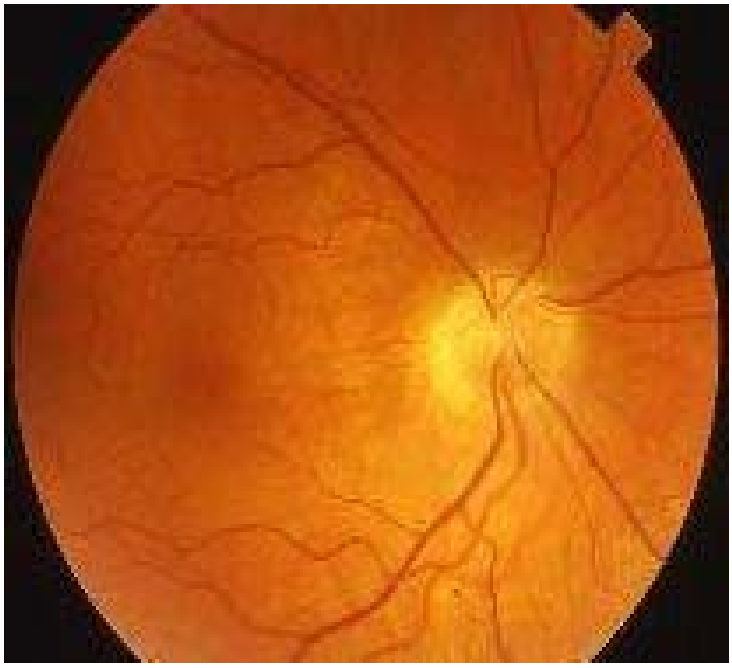
33. Br. J. Ophthalmol, 1964; 48: 522-543, Hayreh ss: pathogenesis of edema of optic disc.

34. Br. J. Ophthalmol, 1980; 64: 211-216, Macular changes resulting from papilledema.

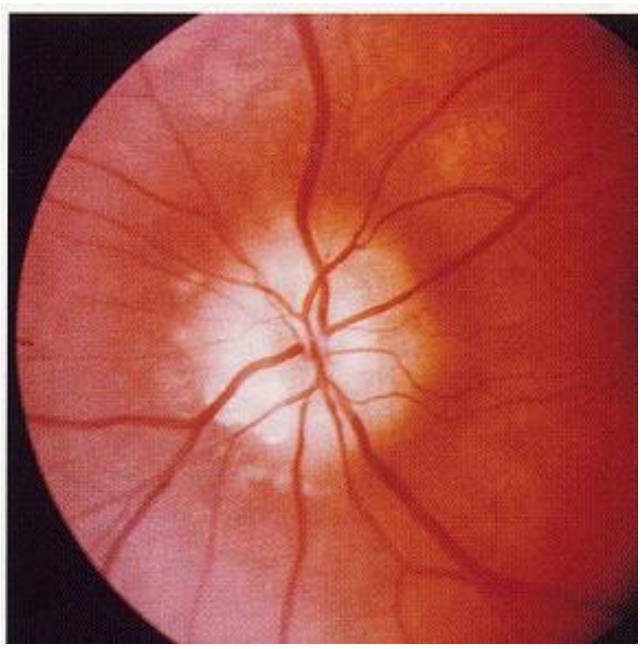
35. Br. J. Ophthalmol, 1973; 57; 89- 97, Choroidal folds in association with papilledema.



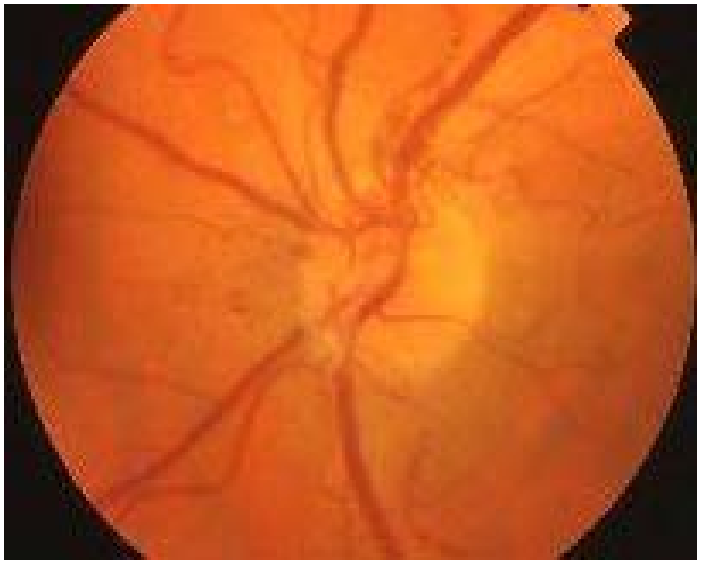
DIABETIC PAPILLOPATHY



NAION

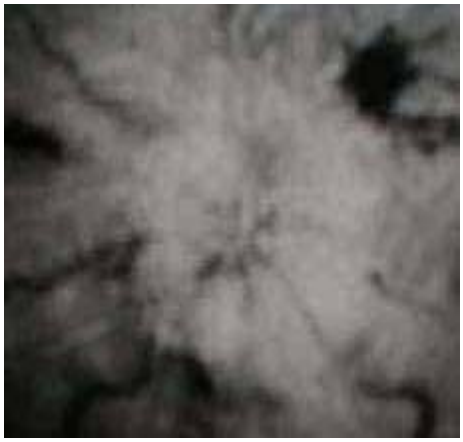


OPTIC DISC DRUSEN



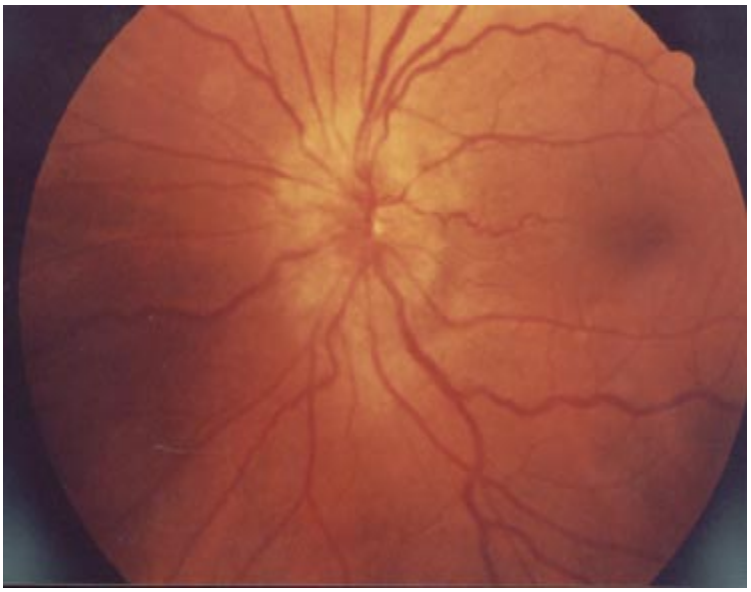
OPTIC NEURITIS

NORMAL FFA PICTURE



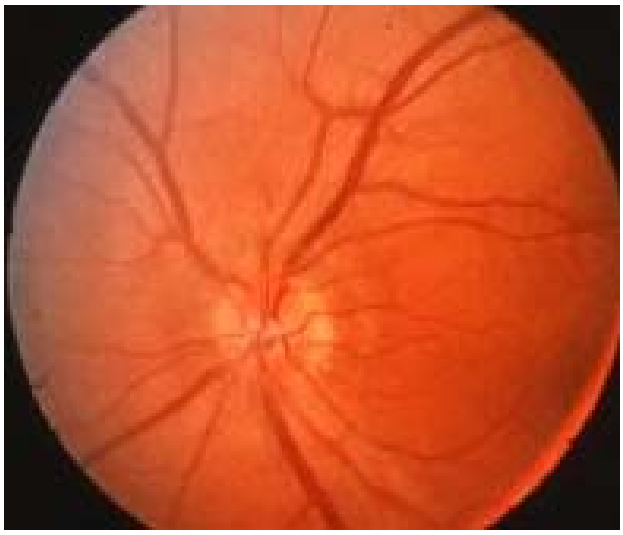
Arteral phase of FFA shows marked dialation of peripapillary capillaries

Venous phase of FFA shows leakage of dye from the vessels

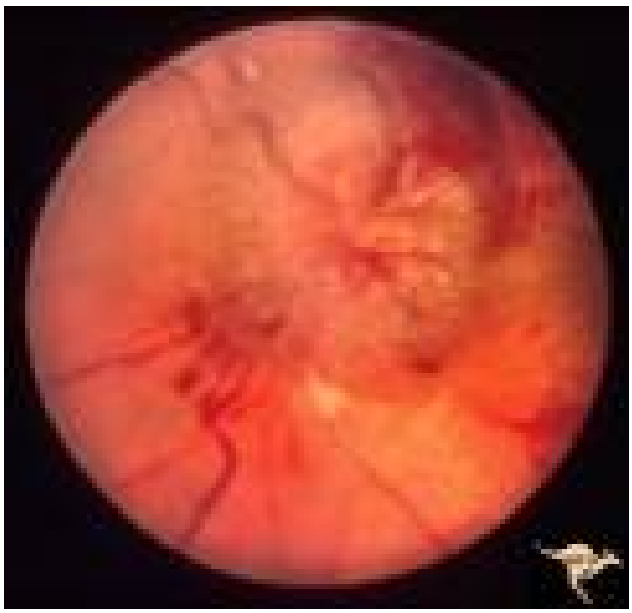


CHRONIC STAGE OF PAPILLDEDMA

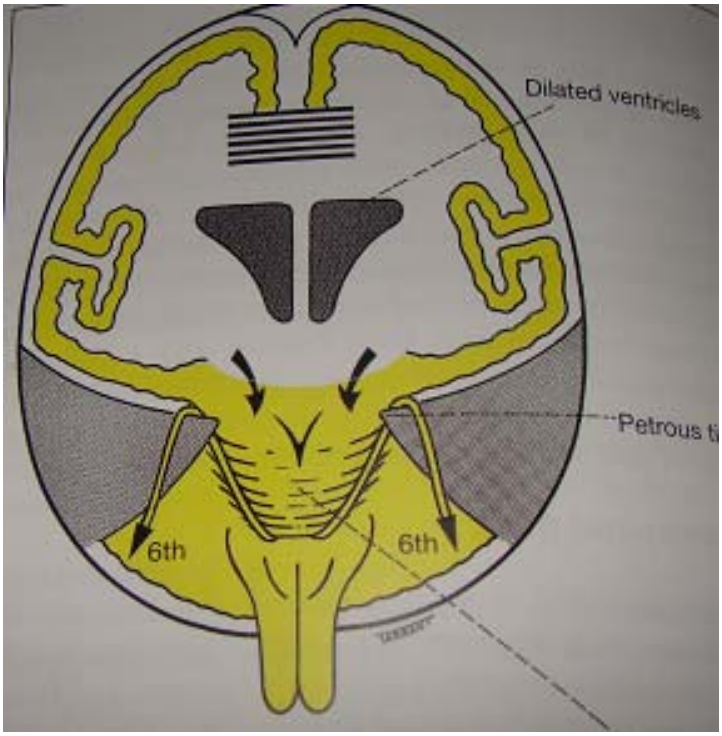
ATROPHIC STAGE OF PAPILLEDEMA



EARLY STAGE OF PAPILLEDEMA



ESTABLISHED STAGE OF PAPILLEDEMA

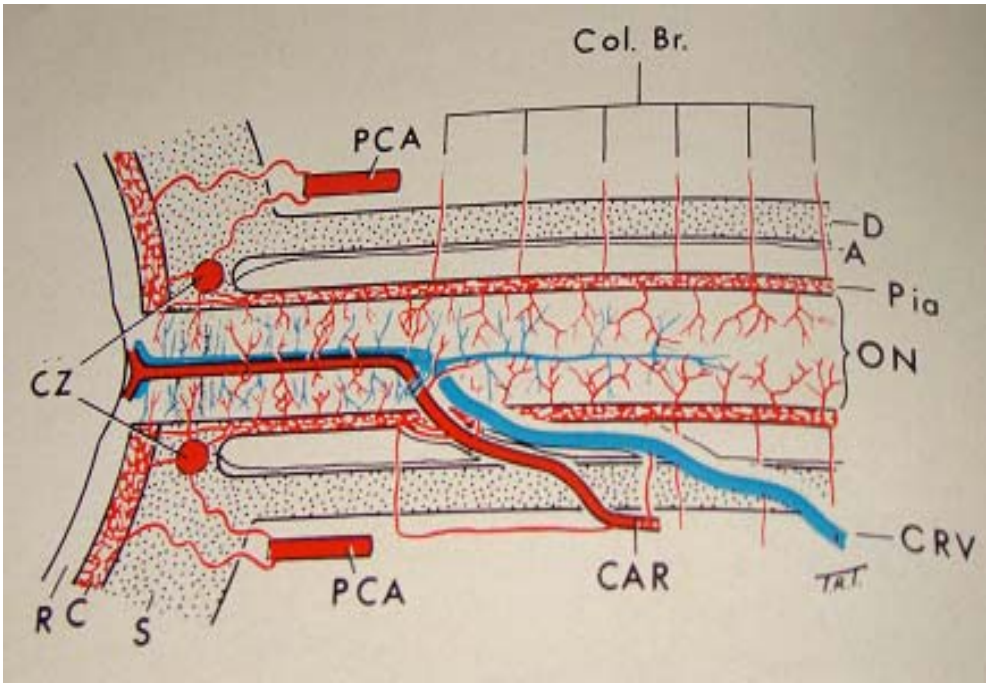


Brain stem pushed downwards

MECHANISM OF 6TH CRANIAL NERVE PALSY FROM

RASIED INTRACRANIL PRESSURE

BLOOD SUPPLY OF OPTIC NERVE[33]



R- Retina

C- Choroid

S- Sclera

PCA- Posterior Ciliary Artery

CZ- Circle of Zinn

Col. Br- Collateral Branches

D- Dura matter

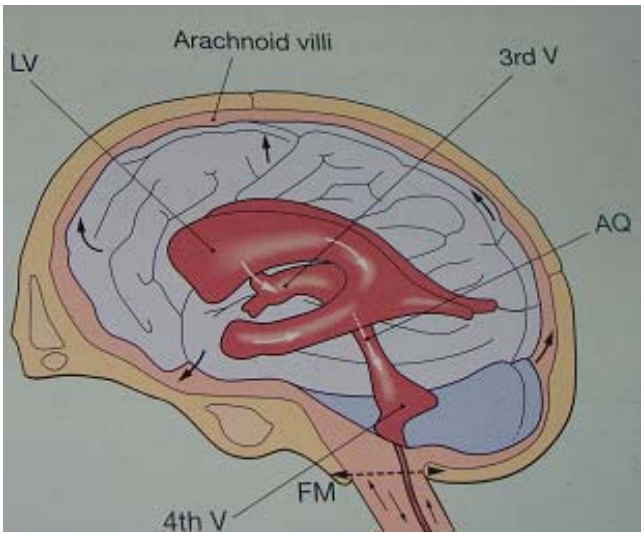
A- Arachnoid matter

Pia- Pia matter

ON- Optic Nerve

CRA- Central Retinal Artery

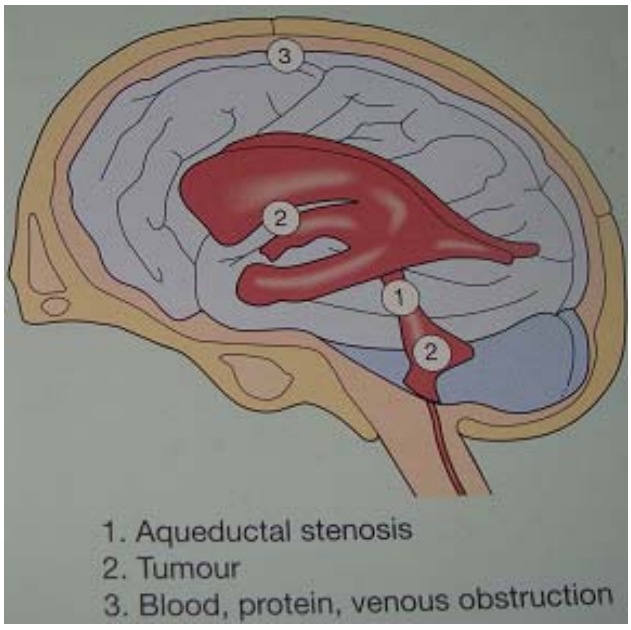
CRV- Central Retina Vein



CIRCULATION OF CEREBROSPINAL FLUID

FM- Foramen Magnum

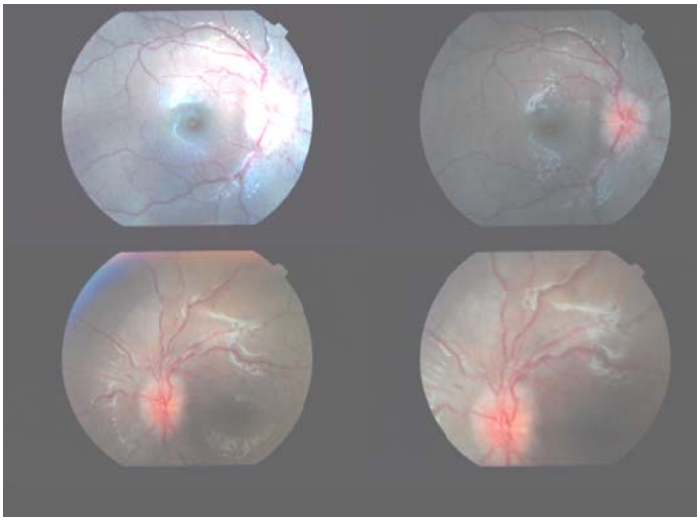
LV- Lateral Ventricle AQ- Acqueduct of silvius

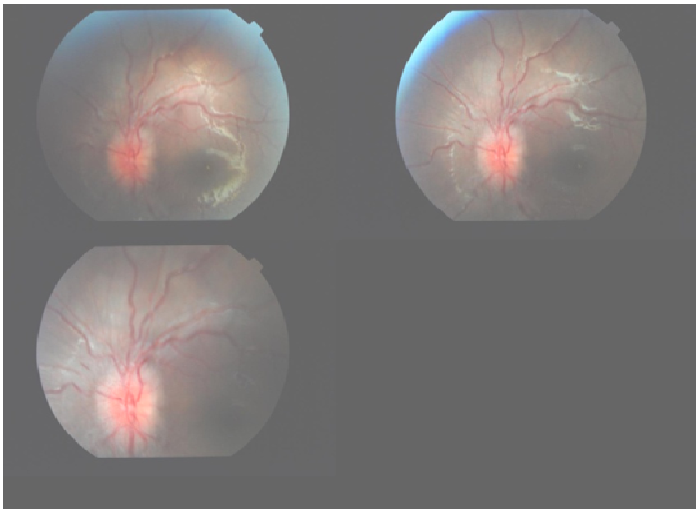


CAUSES OF RAISED INTRACRANIAL PRESSURE

RIGHT SIDE SIXTH CRANIAL NERVE PALSY

CASE NO.7, BE - PAPILLEDEMA





LIST OF SURGERIES

S. No	NAME	AGE/ SEX	IP NO	DIAGNOSIS	PROCEDURE
1.	Sivaprakasam	65/M	382612	RE – MC	RE-ECCE/PCIOL
2.	Leelavathy	67/F	382760	BE–IMC L>R	RE-ECCE/PCIOL
3.	Sivagamy	70/F	332947	BE–MC L>R	LE-ECCE/PCIOL
4.	Rubavathy	60/F	311756	BE–IMC R>L	RE-SICS/PCIOL
5.	Gopal	52/M	391787	BE-IMC R>L	RE-SICS/PCIOL
6.	Krishnan	70/M	392782	LE- PHACOLYTIC GLAUCOMA	RE-ECCE/PCIOL
7.	Savithri	58/F	391999	BE–IMC R>L	RE-SICS/PCIOL
8.	Selvaraj	59/M	396189	LE –IMC	LE-SICS/PCIOL
9.	Elumalai	65/M	399979	RE –IMC LE-PCIOL	RE-SICS/PCIOL
10.	Narayanan	70/M	392156	LE –IMC	LE–SICS/PCIOL
11.	Velusamy	52/M	392436	BE–IMC L>R	RE–SICS/PCIOL
12.	Syed Nazar	65/M	395623	BE-IMC L>R	LE–SICS/PCIOL
13.	Gowri	58/F	395415	LE -MC RE-PCIOL	LE–SICS/PCIOL
14.	John	68/M	394442	BE-IMC R>L	RE–SICS/PCIOL
15.	Ayisha Bee	55/F	356961	BE-IMC R>L	RE–SICS/PCIOL
16.	Devi	65/F	400268	RE –IMC	RE–SICS/PCIOL
17.	Ananthraman	58/M	406474	RE –IMC	RE–SICS/PCIOL
18.	Indrani	56/F	400576	RE –IMC	RE–SICS/PCIOL
19.	Muniammal	50/F	394493	LE – BLEEDING ANTERIOR STAPHYLOMA	LE – EVISCERATION & PMMA

S. No	NAME	AGE/ SEX	IP NO	DIAGNOSIS	PROCEDURE
20.	Ravi	17/M	25789	LE CHALAZION	LE – I & C
21.	Kaveri	37/F	27240	RE-PTERYGIUM	EXCISION WITH AUTO GRAFT
22.	Gopal	55/F	384392	LE-FUNGAL CORNEAL ULCER WITH HYPOPYON	LE – AC WASH WITH AMP – B
23.	Muniappan	59/M	83120	LE-PAN OPHTHALMITIS	LE-EVISCERATION
24.	Shanthi	52/F	376292	RE-ABSOLUTE GLAUCOMA	RE-CYCLOCRYO
25.	Lakshmi	60/F	396867	RE-CHR. DACROCYSTITIS	RE DCT
26.	Babu	59/M	396857	RE- POST OPERATIVE ENDOPHTHALMITIS	RE-INTRA VITREAL INJECTION OF BROAD SPECTRUM ANTBIOTICS
27.	Malakondiah	35/M	387906	RE- DEVELOPMENTAL GLAUCOMA	RE – TRABECULECTOMY
28.	Girija	27/F	402574	RE- SECONDARY GLAUCOMA	RE-AG SURGERY
29.	Shankar	63/M	402574	RE-CHR. DACROCYSTITIS	RE – DCT
30.	Kala	17/F	397265	LE- EXPOSURE KERATOPATHY	LE – TARSORRHAPHY

CHART - 7

VISUAL FIELDS PATTERN IN PAPILLEDEMA

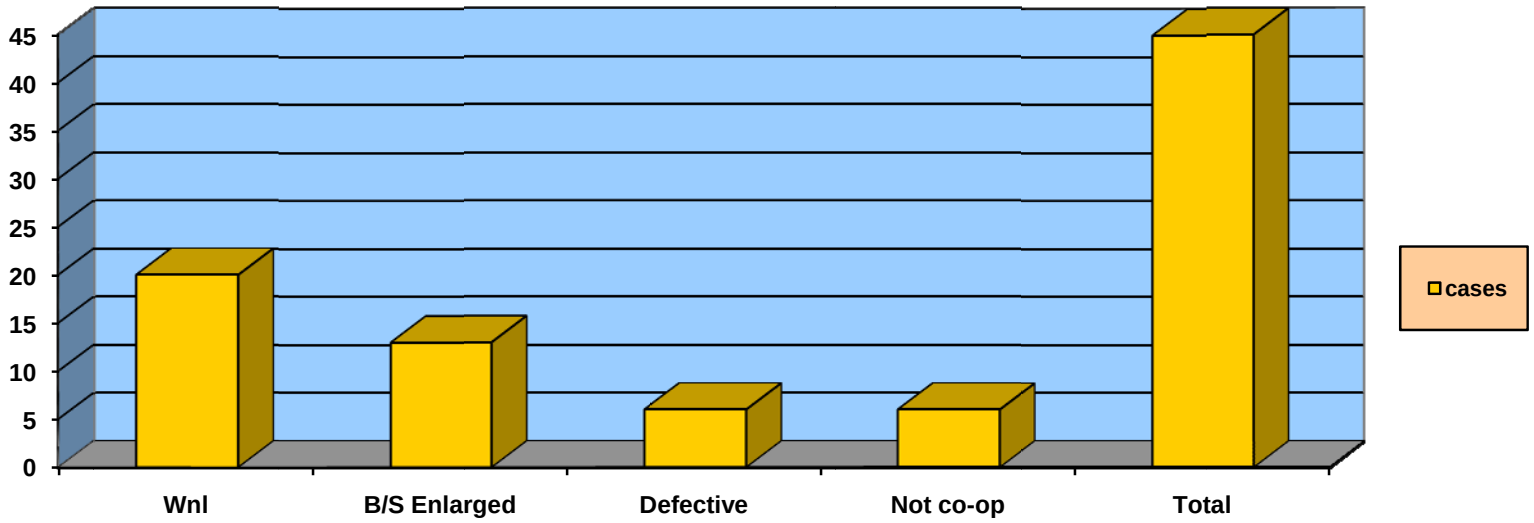


CHART- 6

VISUAL ACUITY PATTERN IN PAPILLEDEMA

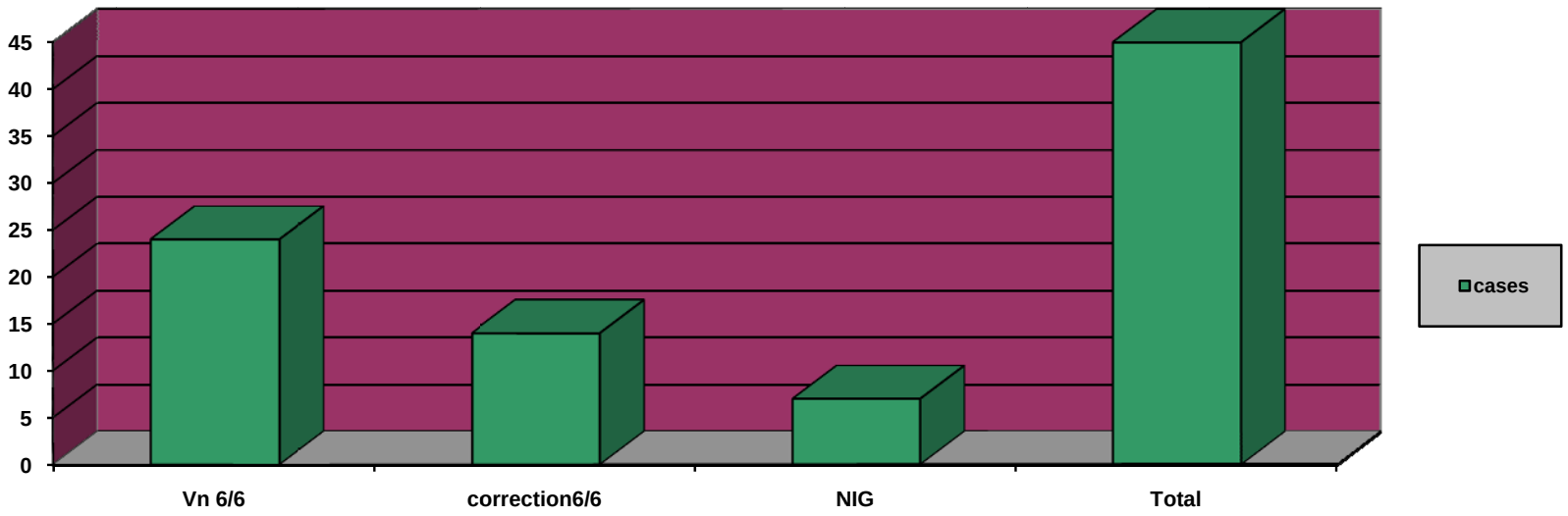


CHART - 5

SIXTH CRANIAL NERVE PALSY IN PAPILLEDEMA

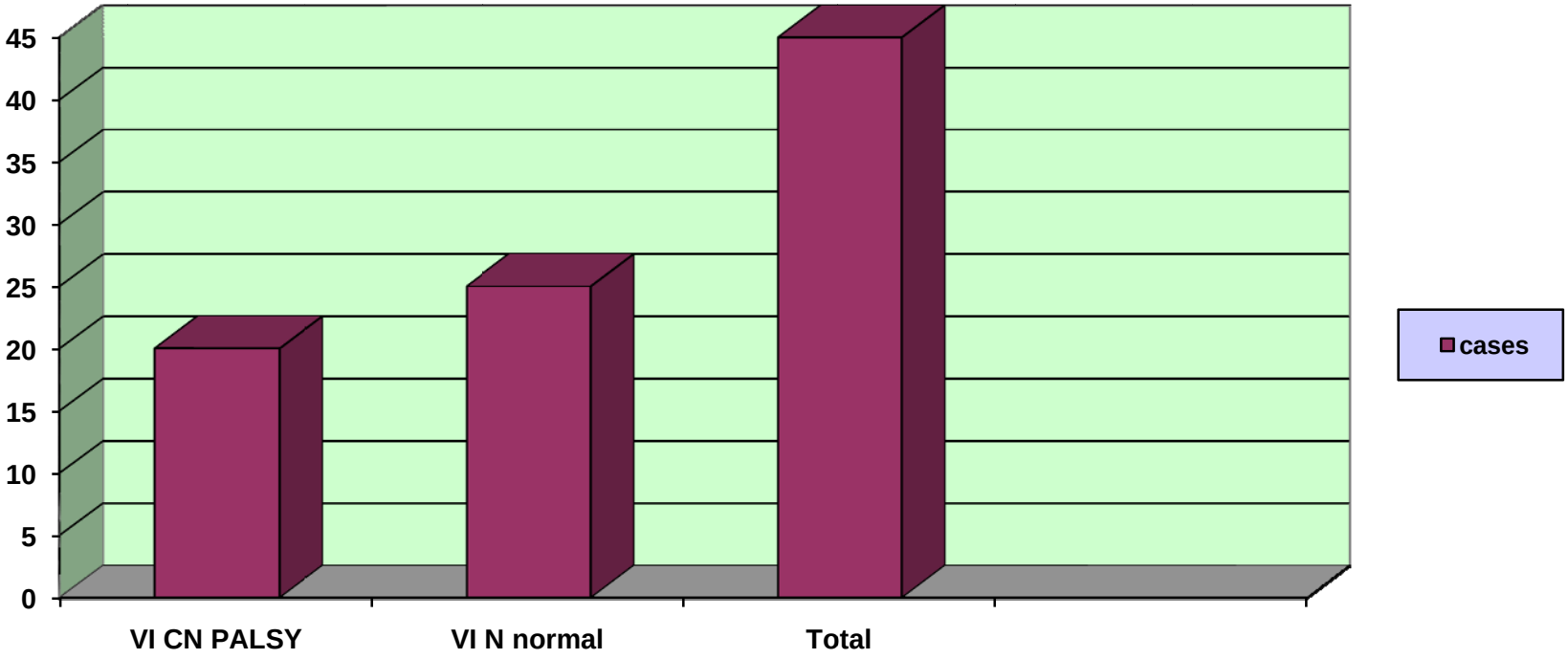


CHART - 4

AETIOLOGICAL PATTERN IN PAPILLEDEMA

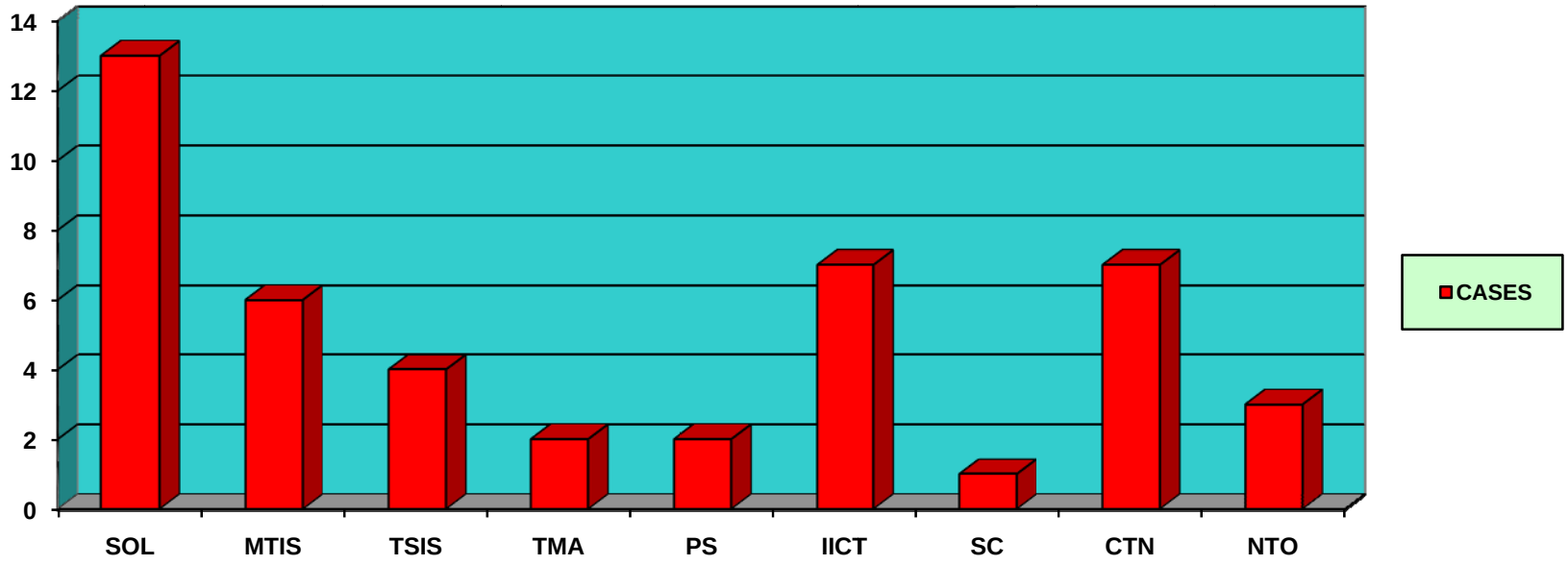


CHART - 3

LATERALITY

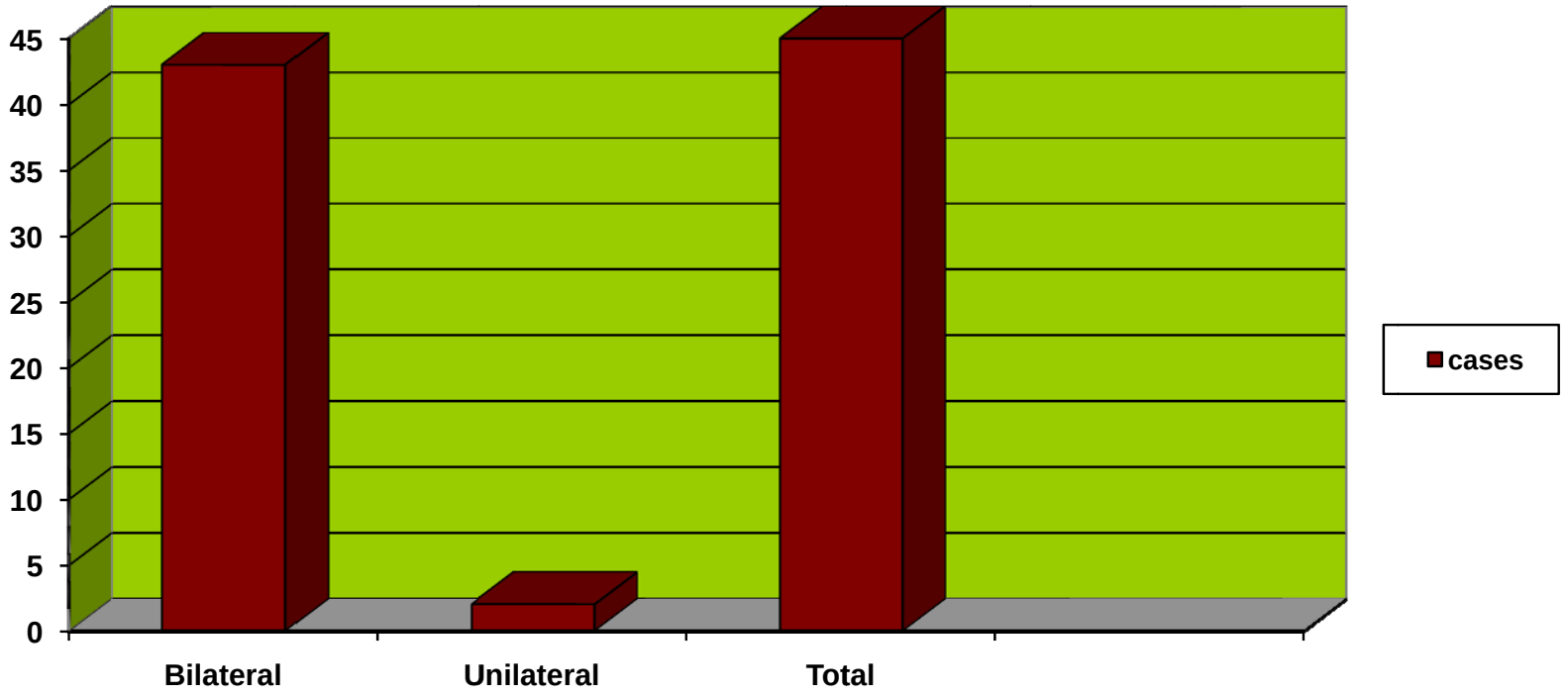


CHART - 2

SEX DISTRIBUTION

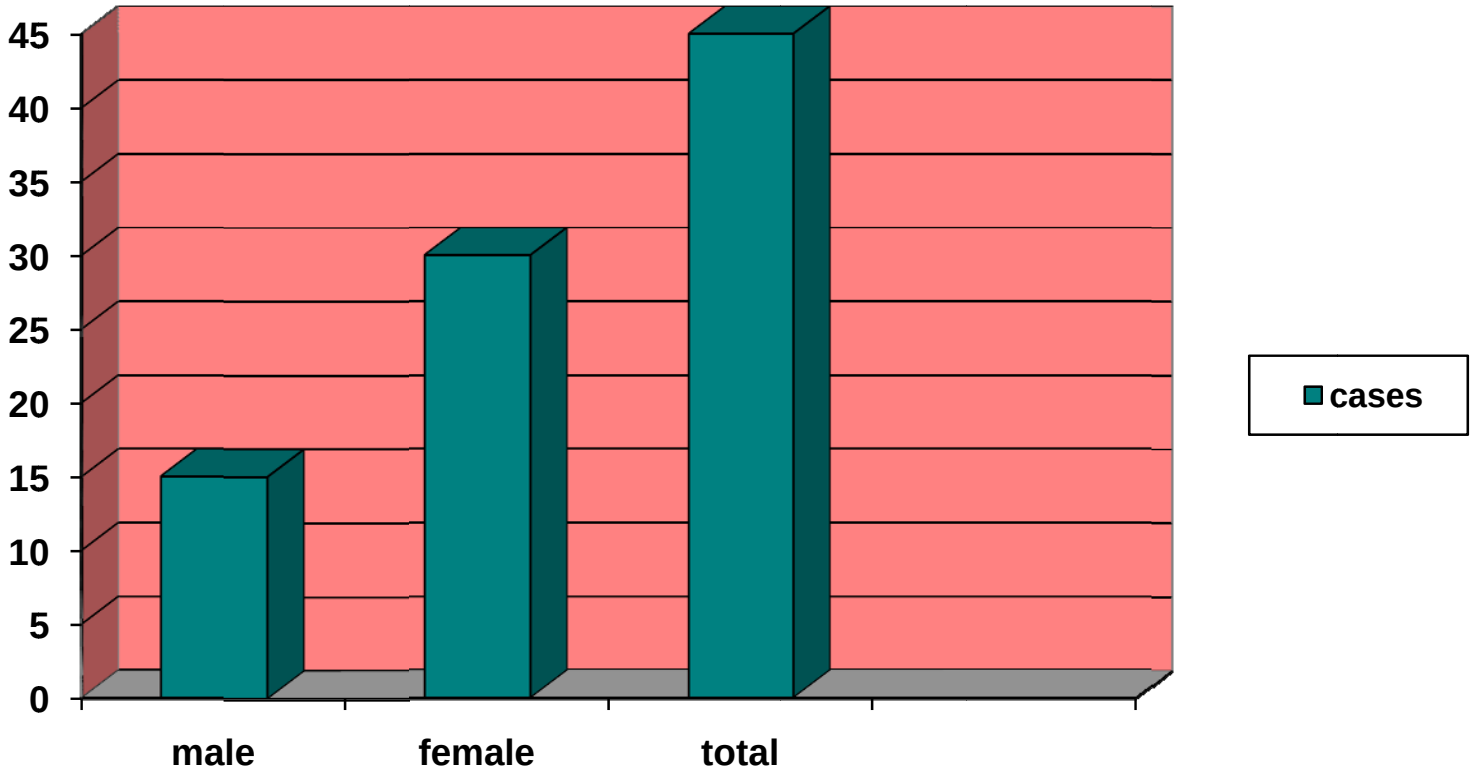
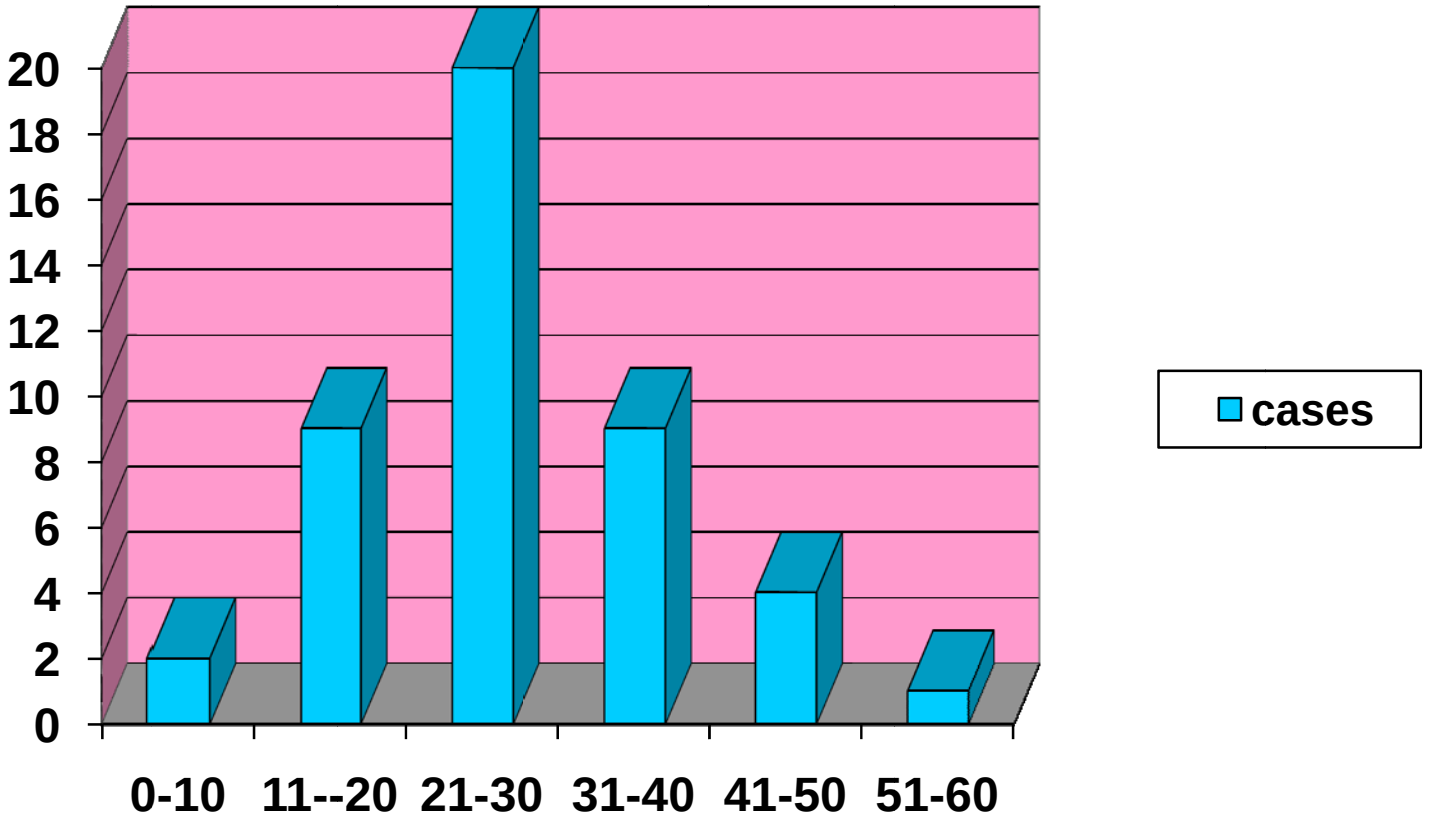


CHART - 1
AGE DISTRIBUTION



PATIENT NAME	AGE	SEX	COMPLAINTS	PUPILL BE	6th CN Palsy	FUNDUS	VISION	Retinoscopy	Correction	Colour Vn BE	FIELDS	TENSION	BP mmHg	CT	MRI/MRV	LP	AETIOLOGY	Sl. No.
Senthamil selvi	26	F	Headache	Normal	Nil	BE Papilledema	BE 6/6P	simple hyperopia	BE +0.5 6/6	Normal	wnl	BE14mmHg	110/80	not done	PPSAS	31cm H2O	IICT	1
Kavitha	26	F	Defective Vn	Normal	Nil	BE Papilledema	6/24 6/18	NIG		Normal	Not co-op	BE14mmHg	100/80	NORMAL			RND	2
Sasi kumar	14	M	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/9	simple myopia	BE _0.5 6/6	Normal	Not co-op	BE 17mmHg	100/70	cerebral edema			TB Meningitis	3
Sangeetha	22	F	Headache/Vomiting	Normal	Present	BE Papilledema	BE 6/6			Normal	Not co-op	BE 17mmHg	130/80	? Cerebral edema			TB Meningitis	4
Sarvanan	29	M	Headache/Vomiting	Normal	Present	BE Papilledema	BE 6/6			Normal	WNL	BE 17mmHg	130/80	B/L SDH			TB/LSDH	5
Nalini	20	F	Headache/pain	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 17mmHg	110/70	not done	BTH		PS	6
Susila	35	F	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/6			Normal	wnl	BE 15mmHg	110/70	NORMAL	Normal		IICT	7
Nagaiya	45	M	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/18	simple hyperopia	BE +I.0 6/6	Normal	Nasal defect BE	BE 15mmHg	140/70	NORMAL	SSST		CVST	8
Krishna moorthy	35	M	Headache/Vomiting	Normal	Present	BE Papilledema	BE 6/6			Normal	wnl	BE 15mmHg	130/90	PMTMS			PMTMS	9
Chitra	29	F	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/12	simple hyperopia	BE +0.75 6/6	Normal	Not reliable	BE 17mmHg	110/70	NORMAL	Normal		IICT	10
Madhu bala	30	F	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 17mmHg	130/80	NORMAL	Normal		IICT	11
Devi	20	F	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 15mmHg	110/80	NORMAL				12
Sulochana	22	F	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 15mmHg	130/70	NORMAL				13
Hari	32	M	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/9	simple hyperopia	BE +0.5 6/6	Normal	B/S Enlarged	BE 16mmHg	130/80	not done	CVST		CVST	14
Maheswari	18	F	Defective Vn	Normal	Nil	BE Papilledema	BE 6/36	Myopia	BE _2.0 6/6	Normal	Not reliable	BE 16mmHg	120/80	MCVST			CVST	15
Selvaganapathy	21	M	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 15mmHg	120/70	not turn off				16
Revathy	22	F	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/6			Normal	wnl	BE 14mmHg	110/70	?NC			NC	17
Ravi	26	M	Double Vn	Normal	Nil	BE Papilledema	BE 6/9	simple hyperopia	BE +0.5 6/6	Normal	wnl	BE 15mmHg	110/70	HLFL			TC	18
Kotteshwari	25	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 16mmHg	110/80	RCMCA			SOL	19
Arunkumar	28	M	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/9	simple hyperopia	BE +0.5 6/6	Normal	wnl	BE 12mmHg	110/70	P3&4V			TB Meningitis	20
Moorthi	29	M	Double Vn	SRTL	Present	BE Papilledema	BE 4/60	NIG		Normal	Defective	BE 17mmHg	110/60	Dilated 3rd &lat.ventricle			AIDS&Meningitis	21
Arulmozhi	12	F	Headache	Normal	Nil	BE Papilledema	BE 6/9	simple hyperopia	BE +0.5 6/6	Normal	wnl	BE 12mmHg	100/60	not done			AML	22
Padmavathy	24	F	Headache/DoubleVn	Normal	Present	BE Papilledema	BE 6/12	simple myopia	BE _0.75 6/6	Normal	Defective	BE 14mmHg	110/70	NORMAL	Normal		APL IgM +ve	23
Maheswari	33	F	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	Contracted	BE 17mmHg	110/80	MIHE			MIHE	24

Kalai arasi	27	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 14mmHg	110/70	DCE	Normal		IICT	25
Deepa lakshmi	7	F	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 12mmHg	110/60	not done			VIT.A overdose	26
Dilli	50	M	Headache	Normal	Nil	BE Papilledema	BE 6/60	NIG		Normal	B/S Enlarged	BE 16mmHg	150/90	not done	?HB		SOL	27
Pushpavathy	45	F	Defective Vn	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 16mmHg	150/90	NORMAL				28
Govindammal	55	F	Diplopia	Normal	Present	BE Papilledema	BE 6/36	NIG		Normal	B/S Enlarged	BE 17mmHg	130/80	GRTCR			SOL	29
Amaravathy	30	F	Defective Vn	SRTL	Present	BE Papilledema	BE 6/18	NIG		Normal	Contracted	BE 16mmHg	120/80	not done	Clival mass		SOL(PS)	30
Bhuvaneshwari	20	F	Defective Vn	Normal	Present	BE Papilledema	BE 6/9	simple myopia	BE _0.5 6/6	Normal	B/S Enlarged	BE 12mmHg	110/70	NORNAL				31
Jaya priya	18	F	Diplopia	Normal	Present	BE Papilledema	BE 6/6			Normal	wnl	BE 13mmHg	100/70	DCE			PMS	32
Suresh	21	M	Diplopia	Normal	Present	BE Papilledema	BE 6/12	NIG		Normal	B/S Enlarged	BE 14mmHg	110/70	not done	CPAEH		NF-II	33
shanthi	36	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	B/S Enlarged	BE 16mmHg	130/90	NORMAL				34
Sivagami	36	F	Defective Vn	Normal	Nil	BE Papilledema	BE 6/12	CM	BE_.5S_.5C6/6	Normal	wnl	BE 16mmHg	120/80	NORMAL				35
Raja	21	M	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 12mmHg	110/70	LCMTE			SOL	36
Rajesh	13	M	Headache/Vomiting	Normal	nil	BE Papilledema	BE 6/6			Normal	wnl	BE 12mmHg		not turn off				37
Siva	28	M	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 13mmHg		not turn off				38
Raja	21	M	Headache/Vomiting	Normal	Present	BE Papilledema	BE 6/6			Normal	wnl	BE 14mmHg	110/70	cerebral edema			TB Meningitis	39
Muthammal	46	F	Defective Vn LE	Normal	Nil	RE Papilledema	6/246/60	RE +1.5S 6/9 LE NIG		Normal	Not co-op	BE 15mmHg	130/80	LOGM			FKS	40
Amudha	33	F	Defective Vn RE	Normal	Nil	LE Papilledema	1/6o6/18	LE +1.00S 6/6 RE NIG		Normal	wnl	BE 16mmHg	120/80	RFLM			FKS	41
Kumary	5	F	Headache/Vomiting	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 12mmHg	100/60	/NC			NC	42
Nirmala	17	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 12mmHg	110/70	pineal mass			pineal mass	43
Banu	31	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	wnl	BE 13mmHg	120/80	NORMAL				44
Sangari	40	F	Headache	Normal	Nil	BE Papilledema	BE 6/6			Normal	HHD	BE 14mmHg	130/80	not done	Empty sella		IICT	45

PROFORMA

PAPILLEDEMA

Name	O.P.NO
Address	I.P.NO
Occupations	Case NO
	Date

COMPLAINTS

Headache / Nausea / Double vision. Transient visual obscuration / visual loss in seconds. Deviation of Eye / Fever / orbital or ocular pain/ Protrusion of Eye / Drooping of eyelid / giddiness / Flashes of light.
Visual disturbances – unilateral / bilateral, Onset, Previous episode, Perception of moving object, Precipitating / Relieving factor.

HISTORY

Fever / Trauma to orbit or head / Weakness / loss of consciousness / Fits / Gait / Sphincter control / Personality disturbances / Bleeding / ENT diseases / Dental caries / Hear diseases / joint pains / Malignancy / Pain & claudication of Jaws.

PAST HISTORY

Hypertension / Diabetes mellitus / Tuberculosis / Syphilis / Exanthematous fever / Connective tissue disorder / Endocrine disorders / Similar episodes / Malignancy / Medical or Surgical or Radiation treatment / Birth history- trauma / Asphyxia / resuscitation / hydrocephalus / normal or forceps delivery.

PERSONAL HISTORY

Exposure to pet animals / Alcohol / Tobacco / Non- Vegetarian / Exposure to STD / Abortion. In female- Obstetric history- Menstrual cycle, APH, PIH, PPH.

FAMILY HISTORY

Similar disease in the family members.

GENERAL EXAMINATION

Pulse B.P Respiratory rate
Temperature
Anemia
Lymphadenopathy
Signs of hyperthyrodism
Focal sepsis

Pedal edema
Cutaneous markers
Congenital anomalies
Mastoid Tenderness

OPHTHALMIC EXAMINATION

Head Posture
Facial Symmetry
Exophthalmos / Enophthalmos RE LE
Eye Position
Eye lids
Ocular movements
Conjunctiva
Cornea
Anterior chamber
Iris
Pupil
- Size
- Shape
- Light reflex
  - Direct
  - Consensual
  - Near reflux

Lens
Anterior vitreous phase Posterior vitreous phase

FUNDUS BOTH DIRECT & INDIRECT

RE LE

Media
Disc
Size
Shape

Margins
Colour
Cup : Disc rato
Lamina cribrosa
Vessels on the disc
Peripapillary region
Edema
Folds
Laminar separation
Hemorrhages
Othres
Vessels
Sheathing
Spontaneous Venous Pulsations
A:V ratio

Periphery
Hemorrhages
Exudates
Others
Macula

WITH 90 D

Fundus Diagnosis
Visual Acuity
Distant

Without correction
With correction
Near
Colour vision
Retinoscopy
Intraocular tension (NCT)

Gonioscopy
Visual fields
Central Peripheral
Diplopia charting
Hess charting

NEUROLOGICAL EXAMINATION

Higher Functions
Cranial Nerves RE LE
Motor System
Sensory System
Cerebellar System
Reflexes
Cardiovascular System, Respiratory System, Gastrointestinal System, Endocrinology System.
E.N.T Examination

PROVISIONAL DIAGNOSIS
INVESTIGATIONS

Hematology :
Hb% TC DC
ESR
RBC count Platelet count
Mantoux Blood VDRL ELISA
Blood sugar- Fasting Post prandial
C.S.F analysis (if any)
Urine – Albumin Sugar Motion – Ova Cyst

Radiological examination:
X – ray Skull lateral view, orbit CT Scan
MRI Scan
Ultrasound Examination
Fundus Fluorescin Angiography

FINAL DIAGNOSIS

Treatment Medical Surgical

FOLLOW UP

Sequelae.

KEY TO MASTER CHART

F	–	Female
M	–	Male
Vn	–	Vision
RE	–	Right Eye
LE	–	Left Eye
BE	–	Both Eye
SRTL	–	Sluggishly Reacting To Light
CN	–	Cranial Nerve
NIG	–	Nil Improvement In Glasses
Wnl	-	With in normal limit
Not co- op	–	Not co- operative
HHD	–	Homonymous Hemianopic Defect
BP	–	Blood Pressure
B/L	–	Bilateral
SDH	–	Sub Dural Hematoma
R, RT	–	Right
CP	–	Cerebello pontine
CVST		Cerebral Venous Sinous Thrombosis
SSST	–	Superior Sagital Sinous Thrombosis
CT	–	Computed Tomography
MRI	–	Magnetic Resonance Imaging
MRV	-	Magnetic Resonance Venography
LP	–	Lumbar Puncture
IICT	–	Idiopathic Intracranial hypertension
TB	–	Tuberculosis
PMTMS	-	Parietal Multiple Tuberculoma with Midline Shift
SOL	–	Space Occupying Lesion
AIDS	–	Acquired Immuno Deficiency Syndrome
AML	–	Acute Myeloblastic Leukemia

APL	–	Anti Phospholipid
CS	–	Compound Myopia
DCE	-	Diffuse Cerebral Edema
RCMCA	-	Right Chronic Mastoiditis with ? Cerebral Abscesses
RND	-	Radical Neck Dissection
T B/L SDH	-	Traumatic Bilateral Sub Dural Hematoma
TC	-	Traumatic Contusions
HB	-	Hemangioblastoma
PS	-	Post Surgical
PMS	-	Post Meningitis Sequlae
NF	-	Neurofibromatosis
PPSAS	-	Prominent Perioptic
MCVST	-	Major Cerebral Venous Sinus Thrombosis
HLFL	-	Hypodense Lesion in Frontal Lobes
MIHE	-	Multiple Intracranial Hemorrhage with Edema
GRTCR	-	Granuloma Right Cerebellar Region
LCMTE	-	Left Chronic Mastoiditis with Temporal Empyema
LOGM	-	Left Olfactory Groove Meningioma
RFLM	-	Right Frontal Lobe Mass
CPAEH	-	Cerebello Pontine Ependymoma with Hydrocephalus
BTH	-	Basilar and Tonsilar Herniation
P3&4V	-	Prominence 3rd & 4th Ventricle
NC	-	Neurocysticercosis
FKS	-	Foster Kennedy Syndrome